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OPTICAL INVESTIGATION OF LIMIT OF VISUAL ACUITY (REPORT 1)

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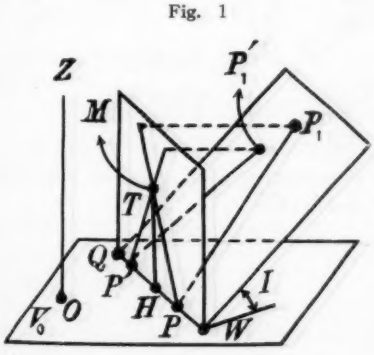
Department of Ophthalmology, School of Medicine, University of Tokyo

In the myopic eye the visual acuity becomes lower as the degree of myopia becomes higher. The present author originated a theory of distribution of saturation and succeeded in deducing the visual acuity from it in a purely theoretical way. The theory covers not only the uncorrected myopic eye but eyes with other kinds of faulty refraction including astigmatism wearing a spherical and a cylindrical lens of any arbitrary strength. He has confirmed that the formula gives values in close agreement with observed values. Report II is devoted to this theory. Report I was added in order to give more rigorous treatment of the case of astigmatic eyes wearing cylindrical lenses.

1. Definition; elementary condition. Let ZO, Fig. 1.1, be the extraocular portion of the visual line, and O be an arbitrarily chosen fixed point on it in front of the cornea, its distance from the cornea being, say, 12 mm.. A plane V_0 passing through O and perpendicular to ZO is termed the initial plane. Draw on V_0 two straight lines XO and YO intersecting at right angles at O so that XO, YO and ZO may constitute a set of rectangular coordinate axes. Let WQ be a straight line on V_0, and, P, a point on WQ. It is now proposed to define two quantities anti-refraction and inclination with respect to the line and the point. Let the distance of P from a fixed point, W on PW be denoted by r, the distance toward Q being considered positive. Let P_1P be the ray of light which passes through P and reaches a point F on the fovea centralis after refraction. Then the angle between the planes V_0 and P_1PQ is termed the inclination at P along PQ and is denoted by $I(r)$. It is to be measured from V_0 to P_1PQ in the counter-clockwise direction seen along PQ in the positive direction. Next consider on PQ another point P' whose distance from W is $r+\Delta r$, and let $P_1'P'$ be the ray passing through P' and reaching F on the fovea centralis. Construct a plane T which intersects V_0 perpendicularly in the line PQ, and draw the projections of PP_1 and $P'P_1'$ on T, letting them meet at M. Denote the height of M above V_0 by ε, considering it positive when M lies away from the eye beyond V_0. The limiting value of the reciprocal of ε as P' approaches P is termed the anti-refraction at P along PQ and is denoted by $A(r)$, that is,

Fig. 1



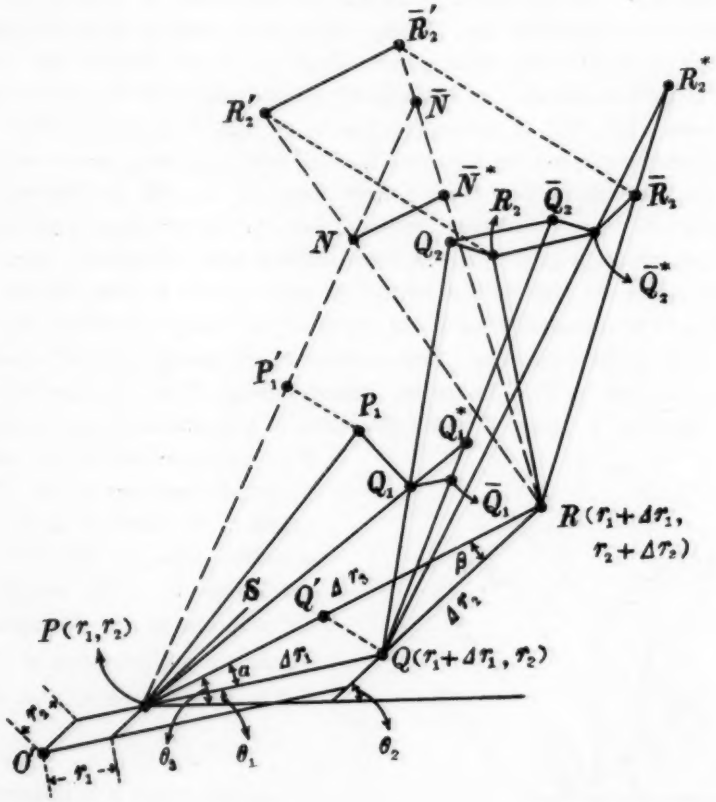
$$A(r)=\lim_{\Delta r\to 0}\frac{1}{\varepsilon} \qquad (1.1)$$

In this report it is assumed that the

anti-refraction, the inclination and the derivative of the latter, $\frac{\partial I(r)}{\partial r}$ are continuous for all values of r whatever the location of PQ on V_0 may be. This assumption is called the elementary condition of anti-refraction and is generally satisfied except when the eye has certain abnormalities such as subluxatio lentis, leucoma corneae and cataracta matura.

2. To find the anti-refraction at any point. Through P (Fig. 1.2) draw any straight lines PQ, PS, PR on V_0. Denote the angles between the X-axis and PQ, PS, PR by $\theta_1,\theta_2,\theta_3$ respectively. Denote the anti-refractions along PQ, PS at P by $A_1(r_1)$, $A_2(r_2)$ respectively, and the inclinations by $I_1(r_1)$, $I_2(r_2)$ respectively. Draw QR parallel to PS meeting PR in R. Let PQ, QR, PR be denoted by Δr_1, Δr_2, Δr_3, respectively. Supposes every one of the rays P_1P, $\bar{Q}_2\bar{Q}_1Q$ and $R_2^*\bar{R}_2R$ comes to F on the fovea centralis. Let $1/A_1(r_1)$, $1/A_2(r_2)$ be denoted by $\varepsilon_1(r_1)$, $\varepsilon_2(r_2)$ respectively. Draw the plane V_1 and the plane V_2 parallel to and at distances $\varepsilon_1(r_1)$, $\varepsilon_2(r_2)$ respectively from V_0. Let P_1P and $\bar{Q}_1Q$ meet V_1 in P_1 and $\bar{Q}_1$ respectively. In general, when r_1 receives an increment Δr_1, $\varepsilon_1(r_1)$ and $I_1(r_1)$ also receive

Fig. 2



increments $\Delta\varepsilon_1$ and ΔI_1 respectively. Now construct on V_0 a set of oblique coordinate axes with O' as origin consisting of an r_1-axis and an r_2- axis parallel to PQ and PS respectively and making an angle $\theta_2-\theta_1$. Then the inclinations along PQ at $P(r_1,r_2)$ and at $Q(r_1+\Delta r_1,r_2)$ may be denoted by $I_1(r_1,r_2)$ and $I_1(r_1+\Delta r_1,r_2)$ respectively. Let $\varepsilon_1(r_1)$ and $\varepsilon_2(r_2)$ at $P(r_1,r_2)$ be denoted by $\varepsilon_1(r_1,r_2)$ and $\varepsilon_2(r_1,r_2)$ respectively. Draw P_1Q_1 on V_1 at right angles to PQ meeting the plane $PQ\bar{Q}_1$ in Q_1. Then

$$P_1Q_1=\varepsilon_1(r_1,r_2)\{\cot I_1(r_1+\Delta r_1,r_2)-\cot I_1(r_1,r_2)\}. \tag{1.2}$$

Produce PQ_1 to intersect $Q\bar{Q}_1$ at Q_1^*. Denote the distance of Q_1^* from V_0 by $\varepsilon_1(r_1,r_2)+\Delta\varepsilon_1'$. When Δr_1 is an infinitesimal $\Delta\varepsilon_1'$ is an infinitesimal, too. (But $\Delta\varepsilon_1'$ is not an increment $\Delta\varepsilon_1$.) Let QQ_1 $Q\bar{Q}_1$, $R\bar{R}_2$ meet V_2 in Q_2, $\bar{Q}_2$, $\bar{R}_2$ respectively. Denote the inclination along QR at R and the inclination along QR at R by $I_2(r_1+\Delta r_1,r_2)$ and $I_2(r_1+\Delta r_1,r_2+\Delta r_2)$ respectively. Draw $\bar{Q}_2\bar{Q}_2^*$ on V_2 at right angles to QR meeting the plane $QR\bar{R}_2$ in $\bar{Q}_2^*$. Then

$$\bar{Q}_2\bar{Q}_2^*=\varepsilon_2(r_1,r_2)\{\cot I_2(r_1+\Delta r_1,\ r_2+\Delta r_2)-\cot I_2(r_1+\Delta r_1,r_2)\}. \tag{1.3}$$

Produce $Q\bar{Q}_2^*$ to intersect $R\bar{R}_2$ at R^*. Denote the distance of R_2^* from V_0 by $\varepsilon_2(r_1,r_2)+\Delta\varepsilon_2'$. When both Δr_1 and Δr_2 approach zero $\Delta\varepsilon_2'$ also approaches zero. Draw Q_2R_2 equal and parallel to $\bar{Q}_2\bar{Q}_2^*$. Through P and R draw a plane T perpendicular to V_0. Draw P_1P_1', QQ', R_2R_2', $\bar{R}_2\bar{R}_2'$ perpendicular to T meeting T in P_1', Q', R_2', $\bar{R}_2'$ respectively. Denote the angle RPQ by α and the angle PRQ by β. That is,

$$\alpha=\theta_3-\theta_1,\ \beta=\theta_2-\theta_3. \tag{1.4}$$

$$\text{Then}\quad R_2\bar{Q}_2^*=Q_2\bar{Q}_2=\frac{\varepsilon_2(r_1,r_2)}{\varepsilon_1(r_1,r_2)}Q_1\bar{Q}_1=\frac{\varepsilon_2(r_1,r_2)}{\varepsilon_1(r_1,r_2)}\cdot\frac{\Delta r_1\cdot\Delta\varepsilon_1'}{\varepsilon_1(r_1,r_2)+\Delta\varepsilon_1'}, \tag{1.5}$$

$$\bar{Q}_2^*\bar{R}_2=\frac{\Delta r_2\cdot\Delta\varepsilon_2'}{\varepsilon_2(r_1,r_2)+\Delta\varepsilon_2'}. \tag{1.6}$$

By (1.5), (1.6),

$$R_2'\bar{R}_2'=R_2\bar{Q}_2^*\cos\alpha+\bar{Q}_2^*\bar{R}_2\cos\beta$$

$$=\frac{\varepsilon_2(r_1,r_2)}{\varepsilon_1(r_1,r_2)}\cdot\frac{\Delta r_1\cdot\Delta\varepsilon_1'\cdot\cos\alpha}{\varepsilon_1(r_1,r_2)+\Delta\varepsilon_1'}+\frac{\Delta r_2\cdot\Delta\varepsilon_2'\cdot\cos\beta}{\varepsilon_2(r_1,r_2)+\Delta\varepsilon_2'}. \tag{1.7}$$

Produce PP_1' to intersect RR_2' and $R\bar{R}_2'$ at N and $\bar{N}$ respectively. Draw $N\bar{N}^*$ parallel to $R_2'\bar{R}_2'$ meeting $R\bar{R}_2'$ in $\bar{N}^*$. Let e and e^* denote the distances of N and $\bar{N}$ respectively from V_0. Then

$$\frac{e}{e^*}=\frac{PN}{P\bar{N}}=\frac{PR-N\bar{N}^*}{PR}=1-\frac{e}{\varepsilon_2(r_1,r_2)}\cdot\frac{R_2'\bar{R}_2'}{PR}. \tag{1.8}$$

From (1.8), by (1.7),

$$\frac{e}{e^*}=1-\frac{e}{\Delta r_1\cos\alpha+\Delta r_2\cos\beta}\cdot\left\{\frac{\cos\alpha}{\varepsilon_1(r_1,r_2)}\cdot\frac{\Delta r_1\cdot\Delta\varepsilon_1'}{\varepsilon_1(r_1,r_2)+\Delta\varepsilon_1'}+\frac{\cos\beta}{\varepsilon_2(r_1,r_2)}\cdot\frac{\Delta r_2\cdot\Delta\varepsilon_2'}{\varepsilon_2(r_1,r_2)+\Delta\varepsilon_2'}\right\}. \tag{1.9}$$

Let $A_3(r_3)$ denote the anti-refraction along PR at P and $\varepsilon_3(r_3)$ denote $1/A_3(r_3)$. Then, when both Δr_1 and Δr_2 approach zero keeping α constant, e^* approaches $\varepsilon_3(r_3)$.

$$\text{From (1.9)},\quad \lim_{\substack{\Delta r_1\to 0\\ \Delta r_2\to 0}}\frac{e}{e^*}=1. \tag{1.10}$$

$$\text{Hence}\quad A_3(r_3)=\lim_{\substack{\Delta r_1\to 0\\ \Delta r_2\to 0}}\frac{1}{e} \tag{1.11}$$

Draw Q_1Q_1' and Q_2Q_2' (Fig. 1.3) perpendicular to T meeting T in Q_1' and Q_2' respec-

tively. Draw $Q'Q_2'$ meeting PP_1' and RR_2' in L and M respectively.

Then $P_1'Q_1'=P_1Q_1 \sin\alpha$.

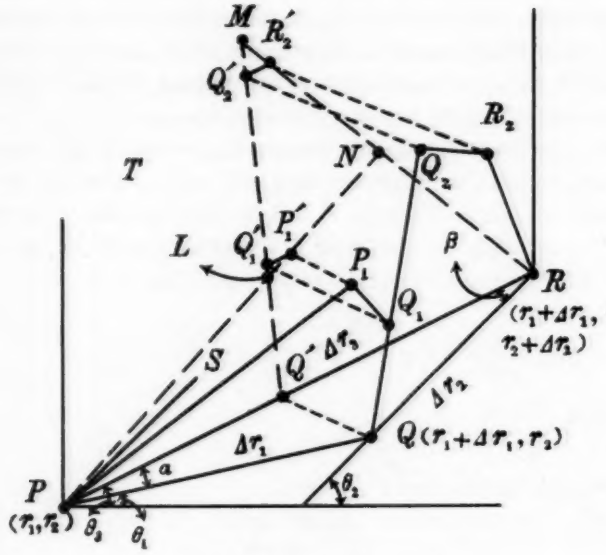
By (1.2) $P_1'Q_1'=\varepsilon_1(r_1,r_2)\{\cot I_1(r_1+\Delta r_1,r_2)-\cot I_1(r_1,r_2)\}\sin\alpha$. (1.12)

And $Q_2'R_2'=Q_2R_2 \sin\beta=\bar{Q}_2\bar{Q}_2^* \sin\beta$.

By (1.3), $Q_2'R_2'=\varepsilon_2(r_1,r_2)\{\cot I_2(r_1+\Delta r_1,r_2+\Delta r_2)-\cot I_2(r_1+\Delta r_1,r_2)\}\sin\beta$. (1.13)

Denote the distances of L and M from V_0 by λ and μ respectively.

Fig. 3



Draw LK (Fig. 1.4) parallel to PR meeting RR_2' in K.

Then $\lambda=\varepsilon_1(r_1,r_2)\dfrac{\Delta r_1\cos\alpha}{\Delta r_1\cos\alpha+P_1'Q_1'}$, (1.14)

$\mu=\varepsilon_2(r_1,r_2)\dfrac{\Delta r_2\cos\beta}{\Delta r_2\cos\beta-Q_2'R_2'}$, (1.15)

$LK=\Delta r_2\cos\beta\cdot\dfrac{\mu-\lambda}{\mu}$, (1.16)

$\dfrac{1}{e}=\dfrac{1}{\lambda}\cdot\dfrac{PR-LK}{PR}=\dfrac{1}{\lambda}\cdot\dfrac{\Delta r_3-LK}{\Delta r_3}$. (1.17)

From (1.17), by (1.14), (1.16), (1.12),

$$\frac{1}{e}=\frac{1}{\varepsilon_1(r_1,r_2)}\cdot\left[1+\frac{\varepsilon_1(r_1,r_2)\{\cot I_1(r_1+\Delta r_1,r_2)-\cot I_1(r_1,r_2)\}\sin\alpha}{\Delta r_1\cos\alpha}\right]\cdot\left[1-\frac{\Delta r_2}{\Delta r_3}\cos\beta\left\{1-\frac{\varepsilon_1(r_1,r_2)\Delta r_1\cos\alpha\cdot(\Delta r_2\cos\beta-Q_2'R_2')}{\varepsilon_2(r_1,r_2)\Delta r_2\cos\beta\cdot(\Delta r_1\cos\alpha+P_1'Q_1')}\right\}\right]. \quad (1.18)$$

From (1.18), by (1.12), (1.13),

$$\frac{1}{e}=\left\{\frac{1}{\varepsilon_1(r_1,r_2)}+\frac{\cot I_1(r_1+\Delta r_1,r_2)-\cot I_1(r_1,r_2)}{\Delta r_1}\cdot\tan\alpha\right\}\cdot\left\{\frac{\Delta r_1}{\Delta r_3}+\frac{\varepsilon_1(r_1,r_2)}{\varepsilon_2(r_1,r_2)}\cdot\frac{\Delta r_2}{\Delta r_3}\right.$$

$$\cos\beta-\varepsilon_2(r_1,r_2)\frac{\cot I_2(r_1+\Delta r_1,r_2+\Delta r_2)-\cot I_2(r_1+\Delta r_1,r_2)}{\Delta r_2}\sin\beta \Big\}\cdot\cos\alpha. \quad (1.19)$$
$$\cos\alpha+\varepsilon_1(r_1,r_2)\frac{\cot I_1(r_1+\Delta r_1,r_2)-\cot I_1(r_1,r_2)}{\Delta r_1}\sin\alpha$$

Now $\dfrac{\Delta r_1}{\sin\beta}=\dfrac{\Delta r_2}{\sin\alpha}=\dfrac{\Delta r_3}{\sin(\alpha+\beta)}$.

From this, $\dfrac{\Delta r_1}{\Delta r_3}=\dfrac{\sin\beta}{\sin(\alpha+\beta)}$; also $\dfrac{\Delta r_2}{\Delta r_3}=\dfrac{\sin(\alpha+\beta)}{\sin\alpha}$. (1.20

From (1.19), by (1.20),

$$\lim_{\substack{\Delta r_1\to 0\\ \Delta r_2\to 0}}\frac{1}{e}=\Big\{\frac{1}{\varepsilon_1(r_1,r_2)}+\frac{\partial\cot I_1(r_1,r_2)}{\partial r_1}\cdot\tan\alpha\cdot\Big\}\cdot\Big\{\frac{\sin\beta}{\sin(\alpha+\beta)}+\frac{\varepsilon_1(r_1,r_2)}{\varepsilon_2(r_1,r_2)}\cdot\frac{\sin\alpha}{\sin(\alpha+\beta)}\cdot$$
$$\frac{\cos\beta-\varepsilon_2(r_1,r_2)\cdot\dfrac{\partial\cot I_2(r_1,r_2)}{\partial r_2}\cdot\sin\beta}{\cos\alpha+\varepsilon_1(r_1,r_2)\cdot\dfrac{\partial\cot I_2(r_1,r_2)}{\partial r_1}\cdot\sin\alpha}\Big\}\cdot\cos\alpha.$$

From this, by (1.11)

$$A_3(r_3)=\Big\{A_1(r_1)+\frac{\partial\cot I_1(r_1,r_2)}{\partial r_1}\cdot\tan\alpha\cdot\Big\}\cdot$$
$$\Big\{\frac{\cos\alpha\sin\beta}{\sin(\alpha+\beta)}+\frac{\sin\alpha\cos\beta}{\sin(\alpha+\beta)}\cdot\frac{A_2(r_2)-\dfrac{\partial\cot I_2(r_1,r_2)}{\partial r_2}\cdot\tan\beta}{A_1(r_1)+\dfrac{\partial\cot I_1(r_1,r_2)}{\partial r_1}\cdot\tan\alpha}\Big\}\cdot$$

Therefore $A_3(r_3)=A_1(r_1)\dfrac{\cos\alpha\sin\beta}{\sin(\alpha+\beta)}+A_2(r_2)\dfrac{\sin\alpha\cos\beta}{\sin(\alpha+\beta)}+U\dfrac{\sin\alpha\sin\beta}{\sin(\alpha+\beta)}$, (1.21)

where $U=\dfrac{\partial\cot I_1(r_1,r_2)}{\partial r_1}-\dfrac{\partial\cot I_2(r_1,r_2)}{\partial r_2}$. (1.22)

If at P $\alpha+\beta=\theta_2-\theta_1=\pi/2$,

then $A_3(r_3)=A_1(r_1)\cos^2\alpha+A_2(r_2)\sin^2\alpha+U\sin\alpha\cos\alpha$. (1.23)

Draw $P\bar{R}$ at right angles to PR on V_0. Let $A_4(r_4)$ denote the anti-refraction along $P\bar{R}$ at P. Then

$$A_4(r_4)=A_1(r_1)\cos^2(\alpha+\frac{\pi}{2})+A_2(r_2)\sin^2(\alpha+\frac{\pi}{2})+U\sin(\alpha+\frac{\pi}{2})\cos(\alpha+\frac{\pi}{2})$$
$$=A_1(r_1)\sin^2\alpha+A_2(r_2)\cos^2\alpha-U\sin\alpha\cos\alpha.$$

$\therefore$ $A_3(r_3)+A_4(r_4)=A_1(r_1)+A_2(r_2)$.

Hence, if $\alpha+\beta=\pi/2$,

$A_1(r_1)+A_2(r_2)=\text{const.}$ (1.24)

Let P be taken as pole. Draw the initial line parallel to the x-axis. Then, using the polar coordinates r,θ, instead of the coordinate r_1,r_2, (1.24) becomes

$A(o,\theta)+A(o,\theta+\frac{\pi}{2})=\text{const.}$ (1.25)

Here $A(o,\theta)$ is the anti-refraction along the radius vector r at P.

Specials case. Erom (1.23), if

$\dfrac{\partial\cot I_1(r_1,r_2)}{\partial r_1}=0$, $\dfrac{\partial\cot I_2(r_1,r_2)}{\partial r_2}=0$, $\alpha+\beta=\dfrac{\pi}{2}$, (1.26)

$A_3(r_3)=A_1(r_1)\cos^2\alpha+A_2(r_2)\sin^2\alpha$. (1.27)

3. To find the inclination at any point. Through P (Fig. 1.5) draw the y-axis at

Fig. 4

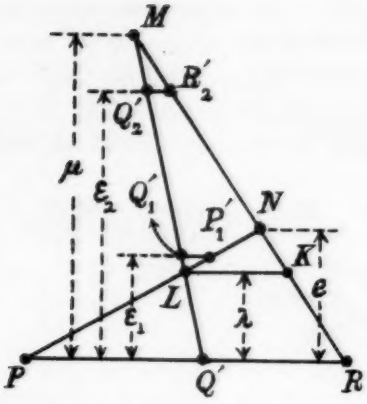
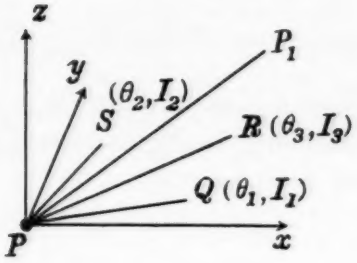


Fig. 5



right angles to the x-axis on V_0 and the z-axis perpendicular to V_0. Let $I_1(r_1)$, $I_2(r_2)$, $I_3(r_3)$ denote the angles between V_0 and the planes P_1PQ, P_1PS, P_1PR respectively. Accordingly $I_1(r_1)$, $I_2(r_2)$ and $I_3(r_3)$ are the inclinations at P along PQ, PS and PR respectively. Let the equation of the plane P_1PQ be

$$l_1x+m_1y+n_1z=0. \quad (l_1^2+m_1^2+n_1^2=1.) \tag{1.28}$$

Then $\cos I_1(r_1)=-n_1.$ (1.29)

The direction cosines of PQ are $\cos\theta_1$, $\sin\theta_1$ and 0. Since PQ lies on the plane P_1PQ,

$$l_1\cos\theta_1+m_1\sin\theta_1=0. \tag{1.30}$$

From (1.28), (1.29), (1.30),

$$l_1=\sin I_1(r_1)\sin\theta_1, \quad m_1=-\sin I_1(r_1)\cos\theta_1, \quad n_1=-\cos I_1(r_1). \tag{1.31}$$

Let the equation of the plane P_1PS be

$$l_2x+m_2y+n_2z=0. \quad (l_2^2+m_2^2+n_2^2=1.) \tag{1.32}$$

Then

$$l_2=\sin I_2(r_2)\sin\theta_2, \quad m_2=-\sin I_2(r_2)\cos\theta_2, \quad n_2=-\cos I_2(r_2). \tag{1.33}$$

P_1P is the intersection of the planes P_1PQ and P_1PS. Accordingly the equations of P_1P are

$$\left.\begin{aligned} x\sin I_1(r_1)\sin\theta_1-y\sin I_1(r_1)\cos\theta_1-z\cos I_1(r_1)=0,\\ x\sin I_2(r_2)\sin\theta_2-y\sin I_2(r_2)\cos\theta_2-z\cos I_2(r_2)=0.\end{aligned}\right\} \tag{1.34}$$

From (1.34), the equations of P_1P are

$$\left.\begin{aligned} \frac{x}{z}&=\frac{\cot I_1(r_1)\cos\theta_2-\cot I_2(r_2)\cos\theta_1}{\sin(\theta_1-\theta_2)},\\ \frac{y}{z}&=\frac{\cot I_1(r_1)\sin\theta_2-\cot I_2(r_2)\sin\theta_1}{\sin(\theta_1-\theta_2)}.\end{aligned}\right\} \tag{1.35}$$

Let the the equation of the plane P_1PR be

$$l_3x+m_3y+n_3z=0. \quad (l_3^2+m_3^2+n_3^2=1.) \tag{1.36}$$

Since PR lies on the plane P_1PR,

$$l_3\cos\theta_3+m_3\sin\theta_3=0. \tag{1.37}$$

From (1.36), by (1.37), the equation of P_1PR is

$$l_3\frac{x}{z}-l_3\frac{y}{z}\cot\theta_3+n_3=0.$$

Now $l_3^2+m_3^2+n_3^2=1$.

From this, by (1.38), $$l_3^2\{1+\cot^2\theta_3+(\frac{x}{z}-\cot\theta_3\frac{y}{z})^2\}=1. \tag{1.39}$$

Put $$B=\frac{1}{l_3^2}, \tag{1.40}$$

Then, since P_1P lies on the plane P_1PR, from (1.39), by (1.35),

$$B=\frac{1}{\sin^2\theta_3}+\frac{[\{\cot I_1(r_1)\cos\theta_2-\cot I_2(r_2)\cos\theta_1\}-\cot\theta_3\{\cot I_1(r_1)\sin\theta_2-\cot I_2(r_2)\sin\theta_1\}]^2}{\sin^2(\theta_1-\theta_2)}. \tag{1.41}$$

Now $l_3=\sin I_3(r_3)\sin\theta_3$.

From this, by (1.40), $\dfrac{1}{\sin^2 I_3(r_3)}=B\sin^2\theta_3$.

$\therefore\ \cot I_3(r_3)=\pm\sqrt{B\sin^2\theta_3-1}$.

From this, by (1.41),

$\cot I_3(r_3)\sin(\theta_1-\theta_2)=\pm\{\cot I_1(r_1)\sin(\theta_3-\theta_2)+\cot I_2(r_2)\sin(\theta_1-\theta_3)\}$.

Hence

$$\cot I_1(r_1)\sin(\theta_2-\theta_3)+\cot I_2(r_2)\sin(\theta_3-\theta_1)+\cot I_3(r_3)\sin(\theta_1-\theta_2)=0. \tag{1.42}$$

When θ_1,θ_2, $I_1(r_1)$, $I_2(r_2)$ and θ_3 are given $I_3(r_3)$ is obtained by (1.42). From (1.42), by (1.4),

$$\cot I_3(r)=\cot I_1(r_1)\frac{\sin\beta}{\sin(\alpha+\beta)}+\cot I_2(r_2)\frac{\sin\alpha}{\sin(\alpha+\beta)}. \tag{1.43}$$

If $\alpha+\beta=\pi/2$,

$$\cot I_3(r_3)=\cot I_1(r_1)\cos\alpha+\cot I_2(r_2)\sin\alpha. \tag{1.44}$$

From this

$$\cot^2 I_3(r_3)=\cot^2 I_1(r_1)\cos^2\alpha+\cot^2 I_2(r)\sin^2\alpha+2\cot I_1(r_1)\cot I_2(r_2)\sin\alpha\cos\alpha.$$

Draw PR perpendicular to PR on V_0. Let $I_4(r_4)$ denote the inclination along $P\bar{R}$ at P. Then

$$\cot^2 I_4(r_4)=\cot^2 I_1(r_1)\sin^2\alpha+\cot^2 I_2(r_2)\cos^2\alpha-2\cot I_1(r_1)\cot I_2(r_2)\cos\alpha\sin\alpha.$$

$\therefore\ \cot^2 I_3(r_3)+\cot^2 I_4(r_4)=\cot^2 I_1(r_1)+\cot^2 I_2(r_2)$

Hence, if $\alpha+\beta=\pi/2$,

$$\cot^2 I_1(r_1)+\cot^2 I_2(r_2)=\text{const.} \tag{1.45}$$

Let P be taken as pole. Draw the initial line parallel to the x-axis. Then, using the polar coordinates r,θ, (1.45) becomes

$$\cot_2 I(0,\theta)+\cot^2 I(0,\theta+\frac{\pi}{2})=\text{const.} \tag{1.46}$$

Here $I(0,\theta)$ is the inclination along the radius vector r at P.

4. To find the derivative of the inclination. In Fig. 1.2, denote the cordinates of P as (r_1,r_3), and the coordinates of R as $(r_1,r_3+\Delta r_3)$. Then the inclinations along PR at P and at R are denoted by $I_3(r_1,r_2)$ and $I_3(r_1,r_3+\Delta r_3)$ respectively. The coordinates of P and the coordinates of R may also be denoted by (r_2,r_3) and $(r_2,r_3+\Delta r_3)$ respectively. Then the inclinations along PR at P and R will be denoted by $I_3(r_2,r_3)$ and $I_3(r_2,r_3+\Delta r_3)$ re-

spectively. In F.g. 1.3,

$$P_1P_1'=\varepsilon_1(r_1,r_2)\cot I_3(r_1,r_2), \tag{1.47}$$

$$Q_1Q_1'=P_1P_1'+P_1Q_1\cos\alpha.$$

From this, by (1.2),

$$Q_1Q_1'=\varepsilon_1(r_1,r_2)\cot I_3(r_1,r_3)+\varepsilon_1(r_1,r_2)\{\cot I_1(r_1+\Delta r_1,r_2)-\cot I_1(r_1,r_2)\}\cos\alpha. \tag{1.48}$$

In $QQ_2Q_2'Q'$, $\dfrac{Q_2Q_2'-QQ'}{Q_1Q_1'-QQ'}=\dfrac{\varepsilon_2(r_1,r_2)}{\varepsilon_1(r_1,r_2)}$.

$$\therefore\quad Q_2Q_2'=\Delta r_1\sin\alpha+\frac{\varepsilon_2(r_1,r_2)}{\varepsilon_1(r_1,r_2)}(Q_1Q_1'-\Delta r_1\sin\alpha). \tag{1.49}$$

In $Q_2R_2R_2'Q_2'$, $R_2R_2'=Q_2Q_2'+Q_2R_2\cos\beta$. (1.50)

Here, by (1.3),

$$Q_2R_2=\bar{Q}_2\bar{Q}_2^*=\varepsilon_2(r_1,r_2)\{\cot I_2(r_1+\Delta r_1,r_2+\Delta r_2)-\cot I_2(r_1+\Delta r_1,r_2)\}.$$

In $R_2\bar{Q}_2^*\bar{R}_2\bar{R}_2'R_2'$, $\bar{R}_2\bar{R}_2'=R_2R_2'+R_2\bar{Q}_2^*\sin\alpha-\bar{Q}_2^*\bar{R}_2\sin\beta$.

From this, by (1.5) and (1.6),

$$\bar{R}_2\bar{R}_2'=R_2R_2'+\frac{\varepsilon_2(r_1,r_2)}{\varepsilon_1(r_1,r_2)}\cdot\frac{\Delta r_1\Delta\varepsilon_1'}{\varepsilon_1(r_1,r_2)+\Delta\varepsilon_1'}\cdot\sin\alpha-\frac{\Delta r_2\Delta\varepsilon_2'}{\varepsilon_2(r_1,r_2)+\Delta\varepsilon_2'}\cdot\sin\beta.$$

$$\therefore\quad\frac{\cot I_3(r_1,r_3+\Delta r_3)-\cot I_3(r_1,r_3)}{\Delta r_3}=\left\{\frac{\bar{R}_2\bar{R}_2'}{\varepsilon_2(r_1,r_2)}-\frac{P_1P_1'}{\varepsilon_1(r_1,r_2)}\right\}\cdot\frac{1}{\Delta r_3}=$$

$$\left\{\frac{Q_2Q_2'+Q_2R_2\cos\beta}{\varepsilon_2(r_1,r_2)}-\cot I_3(r_1,r_2)\right\}\frac{1}{\Delta r_3}$$

$$+\frac{1}{\varepsilon_1(r_1,r_2)}\cdot\frac{\Delta\varepsilon_1'}{\varepsilon_1(r_1,r_2)+\Delta\varepsilon_1'}\cdot\frac{\Delta r_1}{\Delta r_3}\sin\alpha-\frac{1}{\varepsilon_2(r_1,r_2)}\cdot\frac{\Delta\varepsilon_2'}{\varepsilon_1(r_1,r_2)+\Delta\varepsilon_2'}\cdot\frac{\Delta r_2}{\Delta r_3}\cdot\sin\beta=$$

$$\left[\frac{\Delta r_1\sin\alpha}{\varepsilon_2(r_1,r_2)}+\{\cot I_1(r_1+\Delta r_1,r_2)-\cot I_1(r_1,r_2)\}\cos\alpha-\frac{\Delta r_1\sin\alpha}{\varepsilon_1(r_1,r_2)}+\{\cot I_2(r_1+\Delta r,r_2+\Delta r_2)\right.$$

$$\left.-\cot I_2(r_1+\Delta r_1,r_2)\}\cos\beta\right]\cdot\frac{1}{\Delta r_3}+\frac{1}{\varepsilon_1(r_1,r_2)}\cdot$$

$$\frac{\Delta\varepsilon_1'}{\varepsilon_1(r_1,r_2)+\Delta\varepsilon_1'}\cdot\frac{\sin\alpha\sin\beta}{\sin(\alpha+\beta)}-\frac{1}{\varepsilon_2(r_1,r_2)}\cdot\frac{\Delta\varepsilon_2'}{\varepsilon_2(r_1,r_2)+\Delta\varepsilon_2'}\cdot\frac{\sin\alpha\sin\beta}{\sin(\alpha+\beta)}.$$

Hence $\dfrac{\partial\cot I_3(r_1,r_3)}{\partial r_3}=\dfrac{\partial\cot I_1(r_1,r_2)}{\partial r_1}\cdot\dfrac{\cos\alpha\sin\beta}{\sin(\alpha+\beta)}+\dfrac{\partial\cot I_2(r_1,r_2)}{\partial r_2}\cdot\dfrac{\sin\alpha\cos\beta}{\sin(\alpha+\beta)}+$

$$\left\{A_2(r_1,r_2)-A_1(r_1,r_2)\right\}\frac{\sin\alpha\sin\beta}{\sin(\alpha+\beta)}. \tag{1.51}$$

If $\alpha+\beta=\pi/2$,

$$\frac{\partial\cot I_3(r_1,r_3)}{\partial r_3}=\frac{\partial\cot I_1(r_1,r_2)}{\partial r_1}\cos^2\alpha+\frac{\partial\cot I_2(r_1,r_2)}{\partial r_2}$$

$$\sin^2\alpha+\left\{A_2(r_1,r_2)-A_1(r_1,r_2)\right\}\sin\alpha\cos\alpha. \tag{1.52}$$

Draw $P\bar{R}$ perpendicular to PR. Let $I_4(r_1,r_4)$ denote the inclination along $P\bar{R}$ at P. Then

$$\frac{\partial\cot I_4(r_1,r_4)}{\partial r_4}=\frac{\partial\cot I_1(r_1,r_2)}{\partial r_1}\sin^2\alpha+\frac{\partial\cot I_2(r_1,r_2)}{\partial r_2}\cos^2\alpha-\left\{A_2(r_2)-A_1(r_1)\right\}\cos\alpha\sin\alpha.$$

$$\therefore\quad\frac{\partial\cot I_3(r_1,r_3)}{\partial r_3}+\frac{\partial\cot I_4(r_1,r_4)}{\partial r_4}=\frac{\partial\cot I_1(r_1,r_2)}{\partial r_1}+\frac{\partial\cot I_2(r_1,r_2)}{\partial r_2}.$$

Hence, if $\alpha+\beta=\pi/2$,

$$\frac{\partial\cot I_1(r_1,r_2)}{\partial r_1}+\frac{\partial\cot I_2(r_1,r_2)}{\partial r_2}=\text{const.} \tag{1.53}$$

Using the polar coordinates r,θ, as in the preceding case, (1.53) becomes

$$\left[\frac{\partial\cot I(r,\theta)}{\partial r}\right]_{r=0}+\left[\frac{\partial\cot(r,\theta+\frac{\pi}{2})}{\partial r}\right]_{r=0}=\text{const.} \tag{1.54}$$

Here $I(r,\theta)$ is the inclination along the radius vector r at the point (r,θ).

5. To produce the optical effect at any point by decentring and rotating the combination of a thin spherical lens and a thin cylindrical lens. Take an S dioptre (D) sphere and a CD cylinder from the trial case, and place them in contact with each other and with their optical axes common. Let the optical axis meet the combination of the lenses in O, the origin. Through O pass the initial plane V_0 perpendicular to the optical axis. On V_0 (Fig. 1.6), through O draw the u-axis parallel to and the v-axis perpendicular to the axis of the cylinder. Let the strength of this combination along the u-axis be denoted by D_1, and that along the v-axis by D_2. Then

$$D_1=S,\quad D_2=S+C. \tag{1.55}$$

In the optical system of the eye, the anti-refraction and the inclination are defined by the rays coming to the fovea centralis, while, in that of the lenses, they are defined by the rays made parallel to the optical axis by passage through the lenses. Take any point $G(u, v)$ on V_0. Draw GG_1 and GG_2 parallel to the u-axis and the v-axis respectively. On V_0, let GG_3 be drawn so as to make an angle γ with the u-axis. Let $I_1(u)$, $I_2(v)$, $I_3(r_3)$ denote the inclinations along GG_1, GG_2, GG_3 respectively.

$$\left.\begin{aligned}&\text{Then } \cot I_1(u)=vD_2\ ;\\ &\text{also } \cot I_2(v)=-uD_1.\end{aligned}\right\} \tag{1.56}$$

From (1.46), by (1.56),

$$\cot I_3(r_3)=vD_2\cos\gamma-uD_1\sin\gamma. \tag{1.57}$$

Let $A_1(u)$, $A_2(v)$, $A_3(r_3)$ denote the anti-refractions along GG_1, GG_2, GG_3 respectively.

Since $\dfrac{\partial \cot I_1(u,v)}{\partial u}=0$, $\dfrac{\partial \cot I_2(u,v)}{\partial v}=0$,

from (1.27), $A_3(r_3)=D_1\cos^2\gamma+D_2\sin^2\gamma$. (1.58)

Put $\rho=\alpha-\gamma$, (1.59)

in which ρ is a constant.

From (1.25) and (1.58), by (1.59),

$$D_1\cos^2\gamma+D_2\sin^2\gamma=A_1(r_1)\cos^2(\gamma+\rho)+A_2(r_2)\sin^2(\gamma+\rho)+U\sin(\gamma+\rho)\cos(\gamma+\rho). \tag{1.60}$$

Here $U=\dfrac{\partial \cot I_1(r_1,r_2)}{\partial r_1}-\dfrac{\partial \cot I_2(r_1,r_2)}{\partial r_2}$. (1.61)

From (1.46) and (1.57), by (1.59),

$$vD_2\cos\gamma-uD_1\sin\gamma=\cot I_1(r_1)\cos(r+\rho)+\cot I_2(r_2)\sin(\gamma+\rho). \tag{1.62}$$

If (1.60) and (1.62) are satisfied when γ takes any value whatever between $-\pi$ and π, the optical effects at G will be equal to the optical effects at P. From (1.60),

$$D_1\cos^2\gamma+D_2\sin^2\gamma-A_1(r_1)(\cos^2\gamma\cos^2\rho+\sin^2\gamma\sin^2\rho)-A_2(r_2)(\sin^2\gamma\cos^2\rho+\cos^2\gamma\sin^2\rho)+U(\sin^2\gamma-\cos^2\gamma)\sin\rho\cos\rho=[2\{A_2(r_2)-A_1(r_1)\}\cdot\sin\rho\cos\rho+U(\cos^2\rho-\sin^2\rho)]\sin\gamma\cos\gamma. \tag{1.63}$$

From this, $[\{D_1-D_2+A_1(r_1)\sin^2\rho-A_1(r_1)\cos^2\rho+A_2(r_2)\cos^2\rho-A_2(r_2)\sin^2\rho-2U\sin\rho\cos\rho\}^2+\{2(A_2(r_2)-A_1(r_1))\sin\rho\cos\rho+U(\cos^2-\sin^2\rho)^2\}]\cos^4\gamma+[2\{D_2-A_1(r_1)\sin^2\rho-A_2(r_2)\cos^2\rho+U\sin\rho\cos\rho\}\{D_1-D_2+A_1(r_1)\sin^2\rho-A_1(r_1)\cos^2\rho+A_2(r_2)\cos^2\rho-A_2(r_2)\sin^2\rho-2U\sin\rho\cos\rho\}-\{2(A_2(r_2)-A_1(r_1))\sin\rho\cos\rho+U(\cos^2\rho-\sin^2\rho)\}^2]\cos^2\gamma+\{D_2-A_1(r_1)\sin^2\rho-A_2(r_2)\cos^2\rho+U\sin\rho\cos\rho\}=0.$ (1.64)

Since this is satisfied by all values of $\cos^2\gamma$ from 0 to 1, there is obtained:

$$D_2-A_1(r_1)\sin^2\rho-A_2(r_2)\cos^2\rho+U\sin\rho\cos\rho=0, \tag{1.65}$$

$$2\{A_2(r_2)-A_1(r_1)\}\sin\rho\cos\rho+U(\cos^2\rho-\sin^2\rho)=0, \tag{1.66}$$

$$D_1-A_1(r_1)\cos^2\rho-A_2(r_2)\sin^2\rho-U\sin\rho\cos\rho=0. \tag{1.67}$$

From (1.66)

$$4[U^2+\{A_2(r_2)-A_1(r_1)\}^2]\sin^4\rho-4[U^2+\{A_2(r_2)-A_1(r_1)\}^2]\sin^2\rho+U^2=0. \tag{1.68}$$

Put $V=\sqrt{U^2+\{A_2(r_2)-A_1(r_1)\}^2}$. (1.69)

Then on solving (1.68), we get

$$\left.\begin{aligned}\sin\rho&=\pm\sqrt{\frac{1}{2}\pm\frac{A_2(r_2)-A_1(r_1)}{2V}},\\ \cos\rho&=\pm\sqrt{\frac{1}{2}\mp\frac{A_2(r_2)-A_1(r_1)}{2V}},\end{aligned}\right\} \tag{1.70}$$

Substitution of (1.70) in (1.67) and (1.65) gives the values of D_1 and D_2.

If $U\leqq 0$,

$$\left.\begin{aligned}\sin\rho&=\sqrt{\frac{1}{2}-\frac{A_2(r_2)-A_1(r_1)}{2V}},\\ \cos\rho&=\sqrt{\frac{1}{2}+\frac{A_2(r_2)-A_1(r_1)}{2V}},\end{aligned}\right\} \tag{1.71}$$

then $D_1+\frac{1}{2}\{A_1(r_1)+A_2(r_1)\}-\frac{\{A_2(r_2)-A_1(r_1)\}^2}{2V}+\frac{U}{2}\sqrt{1-\frac{\{A_2(r_2)-A_1(r_1)\}^2}{V^2}}$

$=\frac{1}{2}\{A_1(r_1)+A_2(r_2)\}-\frac{\{A_2(r_2)-A_1(r_1)\}^2}{2V}-\frac{1}{2}\cdot\frac{U^2}{V}$.

Accordingly $D_1=\frac{1}{2}\{A_1(r_1)+A_2(r_2)-V\}$. (1.72)

Also $D_2=\frac{1}{2}\{A_1(r_1)+A_2(r_2)\}+\frac{\{A_2(r_2)-A_1(r_1)\}^2}{2V}+\frac{U^2}{2V}=\frac{1}{2}\{A_1(r_1)+A_2(r_2)+V\}$. (1.73)

If $U\geqq 0$,

$$\left.\begin{aligned}\sin\rho&=-\sqrt{\frac{1}{2}-\frac{A_2(r_2)-A_1(r_1)}{2V}},\\ \cos\rho&=\sqrt{\frac{1}{2}+\frac{A_2(r_2)-A_1(r_1)}{2V}},\end{aligned}\right\} \tag{1.74}$$

then $D_1=\frac{1}{2}\{A_1(r_1)+A_2(r_2)-V\}$, $D_2=\frac{1}{2}\{A_1(r_1)+A_2(r_2)+V\}$. (1.75)

It can be casily ascertained that these values of $\sin\rho$, $\cos\rho$, D_1, D_2 satisfy (1.63).

From (1.47), by (1.64), (1.65), (1.67),

$$S=\frac{1}{2}\{A_1(r_1)+A_2(r_2)-V\},\quad C=V. \tag{1.76}$$

From (1.62)

$$\{vD_2-\cot I_1(r_1)\cos\rho-\cot I_2(r_2)\sin\rho\}\cos\gamma=\{uD_1-\cot I_1(r_1)\sin\rho+\cot I_2(r_2)\cos\rho\}\sin\gamma;$$

and $\{vD_2-\cot I_1(r_1)\cos\rho-\cot I_2(r_2)\sin\rho\}^2\cos^2\gamma$

$=\{uD_1-\cot I_1(r_1)\sin\rho+\cot I_2(r_2)\cos\rho\}^2(1-\cos^2\gamma)$. (1.77)

Since (1.77) is satisfied by all values of $\cos^2\gamma$ from 0 to 1, it follows that

$$uD_1-\cot I_1(r_1)\sin\rho+\cot I_2(r_2)\cos\rho=0, \tag{1.78}$$

$$vD_1-\cot I_1(r_1)\cos\rho-\cot I_2(r_2)\sin\rho=0. \tag{1.79}$$

From (1.78) $u=\dfrac{\cot I_1(r_1)\ \sin\rho-\cot I_2(r_2)\ \cos\rho}{D_1}$. (1.80)

From (1.79) $v=\dfrac{\cot I_1(r_1)\ \cos\rho+\cot I_2(r_2)\ \sin\rho}{D_2}$. (1.81)

Hence, on substituting (1.55), (1.71), (1.72), (1.73), (1.74), (1.75) in (1.80), (1.81), we get

$$\left.\begin{aligned} u&=\frac{\cot I_1(r_1)\ \sin\rho-\cot I_2(r_2)\ \cos\rho}{S}\\ v&=\frac{\cot I_1(r_1)\ \cos\rho+\cot I_2(r_2)\ \sin\rho}{S+C}\end{aligned}\right\} \quad (1.82)$$

Accordingly, when the anti-refractions $A_1(r_1)$, $A_2(r_2)$, and the inclinations $(I_1(r_1), I_2(r_2)$ in any two directions, at right angles to each other, at any point P and U (see (1.61)) are given, S,C,ρ, u,v may be obtained. Let the combination rotate about G (Fig. 1.6) through the angle ρ. Then the anti-refraction and the inclination at G in any direction which makes any angle with the axis of the cylinder will be equal in value to the corresponding quantities at P taken in the same direction. The intersection of two planes is only one straight line. Accordingly, since the inclinations at P are respectively equal to the inclinations at G in two or more directions, the two paths of the rays coming to P and G must coincide with each other.

Comparison of the derivatives of the inclinations at P and G. In a direction which makes an angle γ with the axis of the cylinder at G, we have by (1.52),

$$\frac{\partial\cot I_3(r_1,r_3)}{\partial r_3}=(D_2-D_1)\ \sin\gamma\ \cos\gamma=C\ \sin\gamma\ \cos\gamma. \quad (1.83)$$

In the direction which make an angle $\gamma+\rho$ with PQ at P, we have by (1.54),

$$\frac{\partial\cot I_3(r_1,r_3)}{\partial r_3}=\{A_2(r_2)-A_1(r_1)\}\sin(\gamma+\rho)\cos(\gamma+\rho)+\frac{\partial\cot I_1(r_1,r_2)}{\partial r_1}\cos^2(\gamma+\rho)+$$

$$\frac{\partial\cot I_2(r_1,r_2)}{\partial r_2}\sin^2(\gamma+\rho)=\Big[-\{A_2(r_2)-A_1(r_2)\}\sin\rho\cos\rho+\frac{\partial\cot I_1(r_1,r_2}{\partial r_1}\sin^2\rho+$$

$$\frac{\partial\cot I_2(r_1,r_2)}{\partial r_2}\cos^2\rho\Big]\sin^2\gamma+\Big[\{A_2(r_2-A_1(r_1)\}(\cos^2\rho-\sin^2\rho)-2\Big\{\frac{\partial\cot I_2(r_1,r_2)}{\partial r_2}-$$

$$\frac{\partial\cot I_1(r_1,r_2)}{\partial r_1}\Big\}\sin\rho\ \cos\rho\Big]\sin\gamma\cos\gamma+\Big[\{A_2(r_2)-A_1(r_1)\}\sin\rho\ \cos\rho+$$

$$\frac{\partial\cot I_1(r_1,r_2)}{\partial r_1}\cos^2\rho+\frac{\partial\cot I_2(r_1,r_2)}{\partial r_2}\sin^2\rho\Big]\cos^2\gamma. \quad (1.84)$$

Now by (1.71) and (1.74),

$$\sin^2\rho=\frac{1}{2}\Big\{1-\frac{A_2(r_2)-A_1(r_1)}{V}\Big\},\quad \cos^2\rho=\frac{1}{2}\Big\{1+\frac{A_2(r_2)-A_1(r_1)}{V}\Big\}.$$

If $U\leqq 0$,

$$\sin\rho\ \cos\rho=\frac{1}{2}\sqrt{1-\frac{\{A_2(r_2)-A_1(r_1)\}^2}{V^2}}=-\frac{U}{2V},$$

and if $U\geqq 0$,

$$\sin\rho\ \cos\rho=-\frac{1}{2}\sqrt{1-\frac{\{A_2(r_2)-A_1(r_1)\}^2}{V_2}}=-\frac{U}{2V}.$$

On substitution in (1.84),

Fig. 6

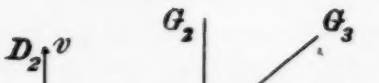
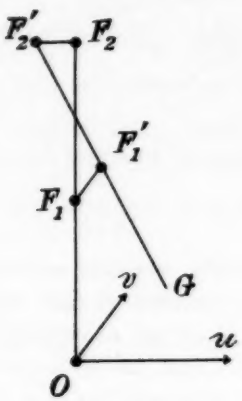


Fig. 7



$$\frac{\partial \cot I_3(r_1,r_3)}{\partial r_3}=\Big[\Big\{A_2(r_2)-A_1(r_1)\Big\}\frac{U}{2V}+\frac{1}{2}\frac{\partial \cot I_1(r_1,r_2)}{\partial r_1}-\frac{A_2(r_2)-A_1(r_1)}{2V}\cdot$$
$$\frac{\partial \cot I_1(r_1,r_2)}{\partial r_1}+\frac{1}{2}\frac{\partial \cot I_2(r_1,r_2)}{\partial r_2}+\frac{A_2(r_2)A_1(r_1)}{2V}\frac{\partial \cot I_2(r_1,r_2)}{\partial r_2}\Big]\sin^2\gamma$$
$$+\Big[\frac{\{A_2(r_2)-A_1(r_1)\}^2}{V}+\frac{U^2}{V}\Big]\sin\gamma\cos\gamma+\Big[-\Big\{A_2(r_2)-A_1(r_1)\Big\}\frac{U}{2V}+\frac{1}{2}\frac{\partial \cot I_1(r_1,r_2)}{\partial r_1}$$
$$+\frac{A_2(r_2)-A_1(r_1)}{2V}\frac{\partial \cot I_1(r_1,r_2)}{\partial r_1}+\frac{1}{2}\frac{\partial \cot I_2(r_1,r_2)}{\partial r_2}-\frac{A_2(r_2)-A_1(r_1)}{2V}\frac{\partial \cot I_2(r_1,r_2)}{\partial r_2}\Big]$$
$$\cos^2\gamma=\frac{1}{2}\Big\{\frac{\partial \cot I_1(r_1,r_2)}{\partial r_1}+\frac{\partial \cot I_2(r_1,r_2)}{\partial r_2}\Big\}\sin^2\gamma+V\sin\gamma\cos\gamma$$
$$+\frac{1}{2}\Big\{\frac{\partial \cot I_1(r_1,r_2)}{\partial r_1}+\frac{\partial \cot I_2(r_1,r_2)}{\partial r_2}\Big\}\cos^2\gamma$$

Hence $$\frac{\partial \cot I_3(r_1,r_3)}{\partial r_3}=\frac{1}{2}\Big\{\frac{\partial \cot I_1(r_1,r_2)}{\partial r_1}+\frac{\partial \cot I_2(r_1,r_2)}{\partial r_2}\Big\}+C\sin\gamma\cos\gamma. \quad (1.85$$

Here, if in (1.53)

$$\frac{\partial \cot I_1(r_1,r_2)}{\partial r_1}+\frac{\partial \cot I_2(r_1,r_1)}{\partial r_2}=0, \quad (1.86)$$

(1.85) becomes equivalent to (1.83).

Using the polar cordinates r, θ (See Art. 4.), (1.86) becomes

$$\Big[\frac{\partial \cot I(r,\theta)}{\partial r}\Big]_{r=0}+\Big[\frac{\partial \cot I(r,\theta+\frac{\pi}{3})}{\partial r}\Big]_{r=0}=0. \quad (1.87)$$

If $\gamma=0$ or $\pi/2$, by (1.83), $\dfrac{\partial \cot I_3(r_1,r_3)}{\partial r_3}=0$.

Hence $\Big[\dfrac{\partial \cot I(r,\theta)}{\partial r}\Big]_{r=0}$ should be zero for one value of θ, say θ_0. That is,

$$\Big[\frac{\partial \cot I(r,\theta)}{\partial r}\Big]_{\substack{r=0,\\ \theta=\theta_0}}=0, \quad \Big[\frac{\partial \cot I(r,\theta)}{\partial r}\Big]_{\substack{r=0,\\ \theta=\theta_0+\frac{\pi}{2}}}=0. \quad (1.88)$$

Rectangular linear foci system. Draw OF_2, Fig. 1.7, perpendicular to the plane u, v. Take the points F_1 and F_2, letting $OF_1=1/D_1$ and $OF_2=1/D_2$. Draw F_1F_1' and F_2F_2' parallel to the v-axis and the u-axis respectively. In order to become parallel to the optical axis OF_2 after passing through the combination of lenses at G a ray must pass

through both the linear foci F_2F_2' and F_1F_1'. Let $F_2'F_1'G$ be the intersection of the planes GF_1F_1' and GF_2F_2'. Only one straight line, i.e. $F_2'F_1'G$ can be drawn through G and meeting F_1F_1' and F_2F_2'. Accordingly the optical system of the combination of the lenses may be defined by the two rectangular linear foci. If (1.86) is satisfied at P, the anti-refraction, the inclination and the derivative of the inclination in any direction at P are equal in value to the corresponding quantities at G taken in the same direction.

Hence the pencil of rays which passes through the small area about P coming to the fovea centralis may approximately be regarded as the rectangular linear foci system. And the two linear foci make the angles θ_0 and $\theta_0+\pi/2$ respectively with the x-axia. (Here $\theta_0=\theta_1+\rho$.)

Summary

Through P draw any straight lines PQ, PS, PR on V_0. Denote the angles made with the x-axis on V_0 by PQ, PS, PR as θ_1, θ_2, θ_3 respectively. Denote the anti-refractions along PQ, PS, PR at P by $A_1(r_1,r_2)$, $A_2(r_1,r_2)$, $A_3(r_1,r_3)$ respectively, and the inclinations by $I_1(r_1,r_2)$, $I_2(r_1,r_2)$, $I_3(r_1,r_3)$ respectively.

Put $\alpha=\theta_3-\theta_1$, $\beta=\theta_2-\theta_3$, (1.4)

$$U=\frac{\partial \cot I_1(r_1,r_2)}{\partial r_1}-\frac{\partial \cot I_2(r_1,r_2)}{\partial r_2}. \tag{1.22}$$

Then $$A_3(r_1,r_3)=A_1(r_1,r_2)\frac{\cos\alpha \sin\beta}{\sin(\alpha+\beta)}+A_2(r_1,r_2)\frac{\sin\alpha\cos\beta}{\sin(\alpha+\beta)}+U\frac{\sin\alpha\sin\beta}{\sin(\alpha+\beta)}, \tag{1.21}$$

$$\cot I_3(r_1,r_3)=\cot I_1(r_1,r_2)\frac{\sin\beta}{\sin(\alpha+\beta)}+\cot I_2(r_1,r_2)\frac{\sin\alpha}{\sin(\alpha+\beta)}. \tag{1.43}$$

$$\frac{\partial \cot I_3(r_1,r_3)}{\partial r_3}=\frac{\partial \cot I_1(r_1,r_2)}{\partial r_1}\frac{\cos\alpha\sin\beta}{\sin(\alpha+\beta)}+\frac{\partial \cot I_2(r_1,r_2)}{\partial r_2}\frac{\sin\alpha\cos\beta}{\sin(\alpha+\beta)}$$
$$+\{A_2(r_1,r_2)-A_1(r_1,r_2)\}\frac{\sin\alpha\sin\beta}{\sin(\alpha+\beta)}. \tag{1.51}$$

Let P be taken as pole. Draw the initial line parallel to the x-axis. Then, using the polar coordinates r, θ, instead of the coordinates r_1,r_2,

$$A(0,\theta)+A(0,\theta+\frac{\pi}{2})=\text{const.} \tag{1.25}$$

$$\cot^2 I(0,\theta)+\cot^2 I(0,\theta+\frac{\pi}{2})=\text{const.} \tag{1.46}$$

$$\left[\frac{\partial \cot I(r,\theta)}{\partial r}\right]_{r=0}+\left[\frac{\partial \cot I(r,\theta+\frac{\pi}{2})}{\partial r}\right]_{r=0}=\text{const.} \tag{1.54}$$

Here $A(0,\theta)$ and $I(0,\theta)$ are respectively the anti-refraction and the inclination along the radius vector r at P, and $I(r,\theta)$ is the inclination along the radius vector r at the point (r,θ).

The anti-refraction and the inclination in any direction at any point P will be equivalent to the corresponding quantities in the same direction at some point G on the combination of some sphere and some cylinder, having a common optical axis, rotated about G through some angles, if these lenses are chosen as follows: Let the anti-refractions at P along any two directions, at right angles to each other, be denoted by $A_1(r_1,r_2)$, $A_2(r_1,r_2)$, and the

inclinations at P along the same two directions by $I_1(r_1,r_2)$, $I_2(r_1,r_2)$. Let the strengths of the sphere and the cylinder be denoted by S and C respectively. Let the optical axis meet the combination in O, the origin. Through O draw the plane V_0 perpendicular to the optical axis. On V_0, draw the u-axis through O parallel to and the v-axis perpendicular to the axis of the cylinder. Denote the coordinates of G about which the combination is rotated by (u, v) and let the angle of rotation be ρ.

Then $$C=\sqrt{U^2+\{A_2(r_1,r_2)-A_1(r_1,r_2)\}^2}, \tag{1.69}$$

$$S=\frac{1}{2}\{A_1(r_1,r_2)+A_2(r_1,r_2)-C\}, \tag{1.76}$$

$$\left.\begin{aligned} u&=\frac{\cot I_1(r_1,r_2)\ \sin\rho-\cot I_2(r_1,r_2)\ \cos\rho}{S},\\ v&=\frac{\cot I_1(r_1,r_2)\ \cos\rho+\cot I_1(r_1,r_2)\ \sin\rho}{S+C}. \end{aligned}\right\} \tag{1.82}$$

Here, if $U\leqq 0$,

$$\sin\rho=\frac{1}{\sqrt{2}}\sqrt{1-\frac{A_2(r_1,r_2)-A_1(r_1,r_2)}{C}},\quad \cos\rho=\frac{1}{\sqrt{2}}\sqrt{1+\frac{A_2(r_1,r_2)-A_1(r_1,r_2)}{C}} \tag{1.71}$$

and if $U\geqq 0$,

$$\sin\rho=-\frac{1}{\sqrt{2}}\sqrt{1-\frac{A_2(r_1,r_2)-A_1(r_1,r_2)}{C}},\quad \cos\rho=\frac{1}{\sqrt{2}}\sqrt{1+\frac{A_2(r_1,r_2)-A_1(r_1,r_2)}{C}}, \tag{1.74}$$

Since the inclinations at P are respectively equal to the inclinations at G in at least two directions, the two paths of the rays coming to P and G must coincide with each other.

If, in (1.54),

$$\left[\frac{\partial \cot I(r,\theta)}{\partial r}\right]_{r=0}+\left[\frac{\partial \cot I(r,\theta+\frac{\pi}{2})}{\partial r}\right]_{r=0}=0, \tag{1.86}$$

P and G have not only equal anti-refraction and inclination but also equal partial derivative of the inclination with respect to r in every direction.

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COMPARISON OF THE HUE DISCRIMINATION BETWEEN BOTH EYES

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The report is very much limited on the unilateral congenital color anomalies and on different type of defectiveness seen between in both eyes. It has been believed in general that the color vision is the same in both eyes except for the cases mentioned above and for the acquired defectiveness. Reviewing the literature, however, it seems likely that

this belief has not yet been proven satisfactorily. Therefore, if there would be a series of cases having different color perception between both eyes, they would give, along with cases of unilateral defectiveness, a good indication of the physiology and heredity of color vision.

We have designed an apparatus which can easily determine the hue discrimination limen. With this apparatus we examined, in each eye of the subject, the discrimination ability of "the most important hue among three attributes of color vision". The subjects were protan, deutan and normal controls. Thus we obtained a considerably different results from the up to data belief. It is therefore, the purpose of the present communication to describe our results and to discuss about the heredity of color defectiveness.

Methods

A modified Nagel's anomaloscope model II was used. The modification was applied on the light source, collimator and exit slit. A light adaptation apparatus was also added to the equipment.

The collimator has two slits, upper and lower one. The width of each slit can be changed by 0.025 mm. with an adjustment screw and scale. The position of the lower slit is fixed (fixed collimator slit). The upper slit can be shifted for 4.0 mm. in each direction from the position of the lower fixed slit (movable collimator slit). A greater movement than 4.0 mm. towards each direction can not give an extention of the examination area in Nagel's anomaloscope. Therefore, there is a limit in the discrimination limen examination; 16.0 mμ at the wave-length of 430 mμ for instance. The deviation of the upper slit from the lower one can be read by 0.005 mm. with a scale. A smoke wedge is inserted behind each collimator slit.

The exit slit is 1.0 mm. in length with the width selection of one of the 0.1, 0.2, 0.3, 0.4 and 0.5 mm.. In the present experiment the width collimator and exit slits is 0.1 mm. for measurement of the wave-length over 480 mμ. The width of collimator slit is 0.25 mm. and that of exit slit 0.2 mm. for measurement of the wave-length below 470 mμ.

The light source is incandescent lamp of 100 V 200 W with a filter of color temperature convert from 2848°K to 6500°K. The voltage is held at 100 V for the measurement of the wave-length below 440 mμ. For the measurement of the wave-length over 450 mμ the brightness of the test field is held constant and appropriate by giving some modifications: the voltage is fixed at 95 V (indicated by F (+) in the tables), or lowered in opposite proportion to the wave-length after a removal of the filter (F(−) in the tables). The visual angle of the object is 2°10′. The light adaptation apparatus, an opaque glass having visual angle 25°, has a brightness 220 rlx illuminated with a standard illuminant C. The relation among the position of collimator slit, grading of the ocular and the wave-length of the exit light is determined by using the line spectrum of Cd (6438, 5086, 4799, 4678 and 4412 Å), Na (5895 Å), and Hg (5790, 5460, 4916, 4358, 4077 and 4047 Å). A calculation table for discrimination limen is thus prepared.

The operation of the apparatus is done by the examiner. The examinee looks the object after a light adaptation and pointed out promptly (within two seconds) whether 1) the upper and lower half of the object look quite the same, 2) one half of the object looks brighter than the other half, or 3) the upper and the lower half look to have the same brightness but they can be differentiated. During the adjustment of the apparatus, the examinee should look at the light adaptation apparatus. Unless the examinee points out No. 3 condition, the examination is repeated by changing the wave-length differently between the upper and the lower half of the object.

When the examinee recognizes the No. 3 condition, the corresponding wave-length difference is regarded as the discrimination limen at the given wave-length of the light. The measurement is repeated at least two or three times; in some cases five times or more. The discrimination limen is measured towards longer and shorter wave at each wave-length of the light. The arithmetic mean is regarded as the final value of the experiment.

The examinee must be trained before the examination. The first step of the training is the measurement of Rayleigh-equation by Hioki's anomaloscope and the second step is a preliminary examination by the present apparatus.

The age and the sex of the examinees are tabulated in tables 1-4. The type of color vision is determined by Ishihara tests for color blindness, AO H-R-R pseudoisochromatic plates and anomaloscope. Acquired color defects are excluded by examination of the anterior ocular segments with ophthalmoscope. The cases in which any acquired ocular diseases are suspected by these examinations are except for a few cases having a slight nebula of the peripheral cornea, and a slight change similar to the persistant pupillary membrane. The color tint of macular pigment is carefully compared between both eyes. No case shows a difference in the macular color tint by ophthalmoscopic examinations. The same care is taken concerning the coloration of the crystalline lens.

Refraction and visual acuity are also examined and only cases with vision of 1.2 and 1.5 are employed. Errors of refraction are corrected, if present, except for slight myopia below 1.0 diopter. Generally, glasses used by the examinees in daily life are used at the time of experiments.

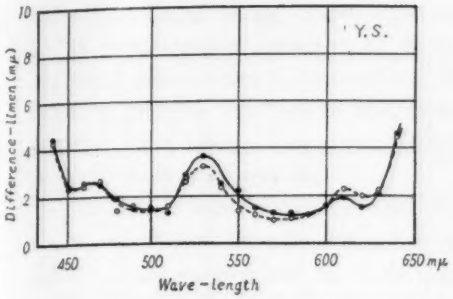
Results

The results are shown in tables 1-4 and fig. 1-6. Only sample cases of the figures are given here owing to a limitation of the given space. The mark (o) at the upper right corner of numbers indicates that, at that wave-length, only the discrimination limen towards longer wave is measurable. The mark (⊿) indicates similarly the limen towards shorter wave is measurable. These limitations are due to the ability of the apparatus itself.

Discussion

In normal persons, protanopia, deuteranopia and protanomalia as shown in tables 1-3 and fig. 1-3, no definite difference in the discrimination limen is recognized between both eyes

Fig. 1

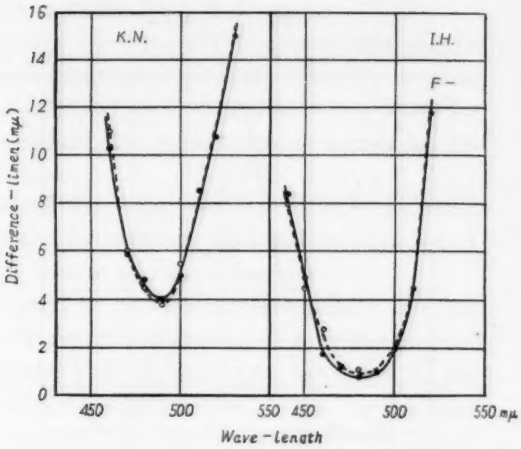


Normal subject (Case 2)

—●— Right eye

—○— Left eye

Fig. 2

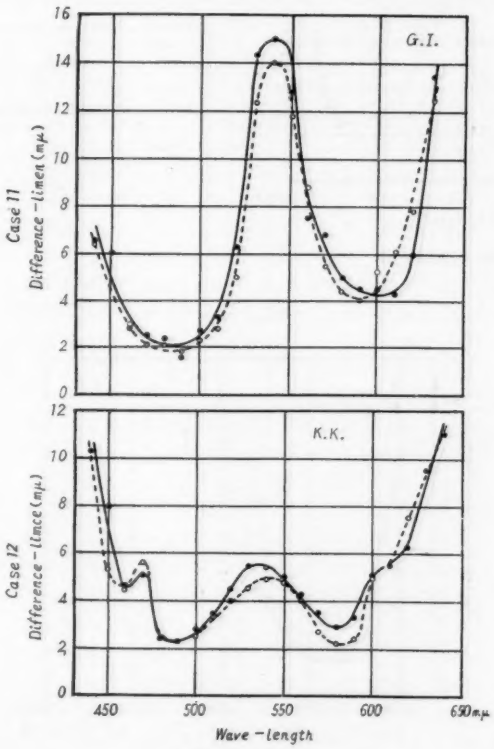


A B

A: Protanopia (Case4)

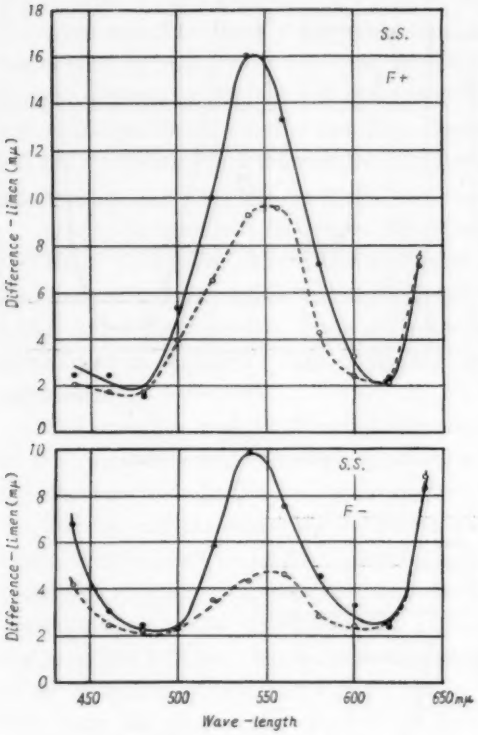
B: Deuteranopia (Case8)

Fig. 3



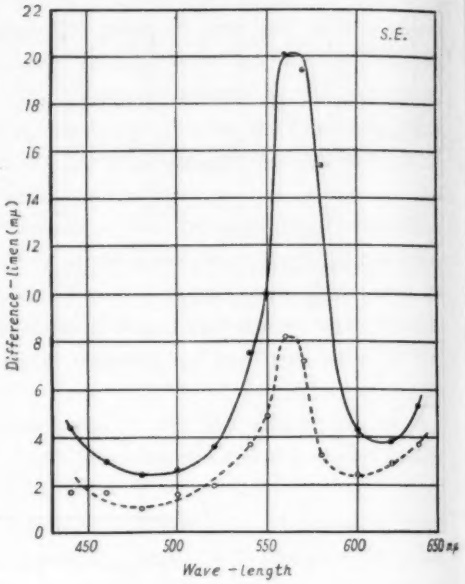
Protanomalis

Fig. 4



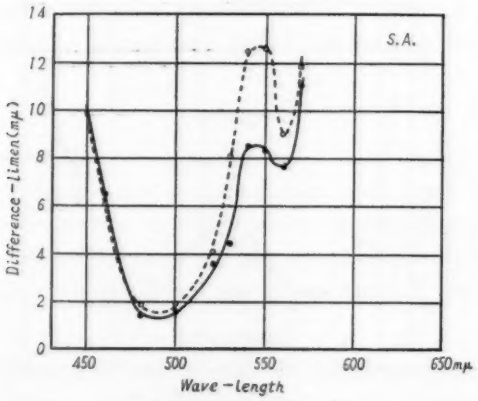
Deuteranomalia (Case 17)

Fig. 5



Deuteranomalia (Case 16)

Fig. 6



Deuteranomalia (Dase 22)

throughout the spectrum. It perfectly coincides with the up to date belief.

In deuteranomalia, however, as shown in table 4 and fig. 4-6, a definite difference in the discrimination limen is demonstrated between both eyes of all cases. Even the smallest difference between both eyes in deuteranomalia is greater than the greatest among differences of other color types. The great difference between both eyes in deuteranomalia can not be an error of measurement.

The measurement is thus repeated on another day. The results are always essentially the same under the same condition. Even under the different condition such as F(+) and F(−), the curve shows the same tendency as shown in fig. 4 and table 4.

When Rayleigh-equation (table 5) is examined, the both eyes of protanomalia give the same equation area. On the contrary, the equation of deuteranomalia, as shown in cases No. 14, 16 and 17, differs from one eye to another; the equation area is greater in the eye of poorer hue discrimination. The most definite example is case No. 16. In this case, the green-yellow equation is demonstrated in the right eye which has poorer hue discrimination, while it is absent in the left eye. It can not be considered to be a measurement

Table 1. Just Noticeable Wavelength Difference of Normal Subjects

Case	1. (S.T. f. 33)				2. (Y.S. f. 28)				3. (K.N. f. 20)			
	F (+)		F (−)		F (+)		F (−)		F (+)		F (−)	
	R	L	R	L	R	L	R	L	R	L	R	L
430 mμ	2.6	2.5	3.5	3.5			6.8	7.0	3.3	3.3	3.9	4.0
440	1.5	1.5	2.0	2.0	4.5	4.3	4.8	5.0	3.1	3.3	3.5	3.0
450	1.4	1.3	1.8	1.9	2.4	2.4	3.1	4.0	1.9	2.4	2.1	2.8
460	0.9	0.8	1.3	1.4	2.6	2.5	2.8	3.0	2.4	2.6	2.0	2.1
470	0.7	0.7	0.8	0.8	2.5	2.6	2.2	2.2	1.1	2.1	1.5	1.5
480	0.7	0.7	0.7	0.8	1.9	1.4	1.5	1.7	0.8	0.8	1.7	1.1
490	0.8	0.8	0.8	0.9	1.5	1.6	2.0	1.8	1.1	1.1	1.6	1.6
500	0.7	1.0	0.9	1.0	1.5	1.5	1.9	1.5	0.9	1.3	2.1	2.3
510	1.1	0.9	1.3	1.1	1.3	1.6	3.0	2.8	1.4	2.0	2.2	2.2
520	1.4	1.8	1.4	1.7	2.9	2.6	4.4	3.5	1.2	1.7	4.2	4.3
530	1.9	1.9	1.9	1.9	3.7	3.3	4.5	3.8	2.0	2.0	4.8	4.5
540	1.8	1.9	1.9	1.9	2.5	2.4	2.8	2.8	1.7	1.7	3.7	3.0
550	1.3	1.5	1.8	1.8	2.2	1.4	2.1	2.1	1.5	1.1	2.3	1.8
560	1.3	1.3	1.7	1.6	1.5	1.2	2.0	2.0	1.2	1.2	1.4	1.4
570	0.7	1.0	1.0	1.3	1.3	1.0	1.5	2.0	1.3	1.3	1.3	1.3
580	0.8	0.8	0.8	1.0	1.3	1.1	1.3	1.8	0.8	1.0	1.1	1.3
590	0.8	0.8	0.8	0.8	1.2	1.2	1.5	2.0	0.8	1.2	0.8	1.5
600	0.8	0.8	0.8	0.8	1.6	1.6	1.9	2.4	1.3	1.3	0.8	1.5
610	1.0	1.0	1.4	1.4	1.9	2.3	2.6	3.0	1.4	1.8	1.8	1.8
620	1.8	1.8	2.3	2.3	1.5	2.0	2.8	3.0	1.8	2.3	3.6	4.0
630	2.0	2.5	3.5	4.5	2.1	2.2	5.1	5.0	1.5	2.5	6.3	6.3
640	3.0	4.3	6.5	8.0	4.6	4.5	8.8	7.6	5.1	4.1	9.3	9.3

Table 2. Just Noticeable Wavelength Difference of Dichromat

Case	Protanopia				Deuteranopia									
	4. (K.N. m. 13)		5. (K.M. m. 23)		6. (K.N. m. 36)		7. (Y.G. m. 24)		8. (I.H. m. 22)				9. (Y.U. m. 10)	
	F (+)		F (−)		F (+)		F (+)		F (+)		F (−)		F (−)	
	R	L	R	L	R	L	R	L	R	L	R	L	R	L
440 mμ			7.6	7.8	15.0°	15.0°	8.6	8.3	8.8°	9.3°	8.4	8.0		
450	12.0°	12.0°	5.5	5.9	6.3	6.3	7.3	7.5	5.3	6.3	4.9	4.5	11.0	11.5
460	10.3	11.0	2.1	1.8	6.0	5.5	5.9	6.0	2.9	2.8	1.7	2.8	5.4	5.5
470	5.9	6.0	1.9	1.3	4.8	4.6	3.6	3.3	1.5	1.4	1.2	1.2	2.4	2.1
480	4.8	4.5	1.2	0.8	3.3	3.3	2.9	1.5	1.4	1.3	0.8	1.1	1.8	2.0
490	4.0	3.8	1.1	1.3	2.5	3.3	2.3	2.0	2.0	1.5	1.0	1.0	2.3	2.3
500	5.0	5.5	1.8	1.8	5.8	7.5	2.8	2.6	3.8	2.5	2.0	2.0	4.0	4.0
510	8.5	8.5	2.3	2.0	8.6	8.8	7.0	7.0	7.9	8.0	4.5	4.5	8.0	8.5
520	10.8	10.8	16.5	22.0	21.8	22.5	18.3	18.4	16.5⊿	26.3⊿	11.8	11.8	21.0	21.0
530	15.0	15.0												

Table 3. Just Noticeable Wavelength Difference of Protanomalia

Case	10. (S.I. m. 27)		11. (G.I. m. 29)		12. (K.K. f. 26)		13. (H.H. m. 18)	
	F (+)		F (−)		F (−)		F (−)	
	R	L	R	L	R	L	R	L
430 mμ	4.0	4.9						
440	2.3	2.0	6.5	6.4	10.3	10.3	1.4	1.3
450	0.9	1.4	6.0	6.0	8.0	5.3		
460	0.4	1.6	2.8	2.9	4.6	4.5	1.3	1.3
470	0.9	0.8	2.5	2.1	5.1	5.6		
480	0.9	0.8	2.4	2.1	2.4	2.4	1.1	0.9
490	0.8	1.0	1.6	1.8	2.3	2.3		
500	0.8	0.8	2.7	2·3	2.8	2.5	1.8	1.2
510	0.8	0.8	3.3	2.8	3.5	3.4		
520	1.7	1.6	6.3	5.0	4.5	4.0	2.0	1.8
530	2.1	2.1	14.3	12.3	5.5	4.5	1.9	2.1
540	2.8	2.3	15.0	14.0	5.4	4.9	2.2	2.0
550	3.1	2.6	12.8	11.8	5.0	4.8	2.7	2.4
560	3.3	2.1	7.5	8.8	4.3	4.0	3.0	2.3
570	2.7	1.9	6.8	5.5	3.5	2.7		
580	1.5	1.2	5.0	4.4	2.9	2.2	2.0	1.8
590	1.7	1.2	4.5	4.0	3.3	2.4		
600	1.7	1.4	4.5	5.3	5.1	5.0	1.6	1.6
610	1.8	1.8	4.3	6.1	5.5	5.5		
620	1.8	3.1	6.0	7.8	6.3	7.5	1.9	1.4
630	4.9	3.0	13.5	12.5	9.5	9.5		
640	12.5	10.5			11.0	11.0	2.9	2.4

Table 4. Just Noticeable Wavelength Difference of Deuteranomalia

Case	14. (M.S. m. 34)		15. (H.U. m. 19)		16. (S.E. m. 48)		17. (S.S. m. 25)				18. (Y.E. f. 16)				19. (G.O. m. 28)		20. (K.K. m. 48)		21. (H.U. f. 47)		22. (S.A. m. 20)		23. (H.O. f. 20)		24. (M.W. f. 17)	
	F(+)		F(+)		F(+)		F(+)		F(−)		F(+)		F(−)		F(−)		F(−)		F(−)		F(−)		F(−)		F(−)	
	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L
430 mμ											5.6	8.0														
440	8.0	4.8	14.0	4.5	4.4	1.7	2.5	2.1	6.8	4.2	3.0	7.5	5.8	4.5	6.8	4.8	13.5°	11.8°	13.5	9.0	10.5	13.0	6.1	4.8	2.3	2.6
450	5.8	4.4	10.0	5.0							3.1	5.8	3.1	4.0	6.0	4.4										
460	3.9	3.3	7.3	3.1	3.0	1.7	2.5	1.8	3.0	2.4	1.9	2.8	1.9	2.1	3.0	2.4	9.0	8.0	4.6	2.8	6.5	6.1	1.8	1.8	2.3	2.7
470	2.5	2.4	3.6	1.5							2.0	2.8	0.9		1.9	1.9										
480	2.0	2.3	2.3	0.6	2.4	1.0	1.6	1.8	2.4	2.2	0.6	1.8	0.9	0.9	1.3	1.3	4.5	3.8	2.8	2.2	1.4	1.9	1.6	1.2	3.1	2.7
490	1.4	1.5	2.8	2.0							0.7	1.6	1.5		1.5	1.5										
500	1.3	1.4	2.3	2.3	2.6	1.6	5.4	4.0	2.3	2.3	1.8	2.1	2.8	1.6	1.6	1.3	5.3	4.0	2.5	2.8	1.6	1.9	2.5	1.3	2.5	2.1
510	1.8	1.3	4.3	3.3							1.8	2.0	3.0		2.5	1.4										
520	2.0	2.3	9.3	9.4	3.6	2.0	10.0	6.5	5.8	3.5	6.3	3.3	4.8	2.8	3.9	2.4	6.8	5.0	2.5	2.8	3.6	4.1	3.4	2.9	12.1	6.6
530	6.5	4.5	13.5	10.0							6.8	3.7	5.5		4.6	2.7					4.4	8.1			14.2	6.7
540	8.8	5.0	13.9	23.6	7.5	3.7	16.0	9.3	9.8	4.3	8.5	3.9	6.8	3.8	7.3	5.0	8.9	6.4	3.3	4.3	8.5	12.5	7.0	3.8	10.3	7.0
550	12.5	5.8	16.8	18.8	10.0	4.9					4.3	3.3	6.9		10.8	5.8					8.3	12.6			8.9	5.3
560	11.0	6.9	17.3	14.0	20.1	8.2	13.3	9.6	7.5	4.6	4.3	2.6	4.6	3.5	10.0	5.5	11.8	9.6	4.0	5.8	7.6	9.0	4.2	4.0	3.9	2.9
570	9.0	5.9	10.1	15.9	19.4	7.2					2.9	2.2	2.9		5.0	5.0					11.0	11.8				
580	7.3	5.5	9.3	9.6	15.4	3.2	7.2	4.3	4.8	2.8	2.2	1.8	2.1	2.2	3.6	3.8	20.5	17.0	3.0	4.0	15.5⊿	16.5⊿	2.7	2.2	4.2	3.3
590	3.9	3.8	6.3	4.0							3.1	1.5	3.1		2.4	2.4										
600	4.3	3.4	3.3	3.4	4.3	2.4	3.3	2.5	3.3	2.4	3.3	1.5	3.3	1.6	2.4	2.4	17.0	15.5	2.9	2.6	33.5⊿	33.5⊿	3.7	4.6	2.8	1.9
610	3.8	3.4	2.6	3.4							3.4	2.2	3.9		2.6	2.0										
620	3.8	3.8	2.5	3.6	3.8	2.9	2.4	2.2	2.4	2.5	4.5	2.8	4.0	2.8	2.8	4.1	13.0	13.3	4.6	4.3	7.5⊿	7.5⊿	6.5	4.5	2.8	2.0
630	4.0	4.0	5.0	5.0							5.0	5.0	4.0		3.0	3.0										
640	5.3	5.3	4.1	6.1	5.3	3.7	7.1	7.4	8.3	8.8	12.8	7.1	11.5	4.6	10.8	10.8	24.8	19.0	8.8	6.8			8.5	8.8	2.9	2.4

Table 5. Rayleigh-equation of Anomalous Trichromat by Hioki's Anomaloscope

Case		Rayleigh-equation
10	R	60.0—80.0
	L	62.0—80.0
11	R	60.0—70.0
	L	60.0—75.0
12	R	58.0—65.0
	L	58.0—62.0
13	R	71.0—75.0
	L	70.0
14	R	5.0—27.0
	L	26.0—33.0
15	R	27.0—41.0
	L	25.0—45.0
16	R	0 —50.0
	L	15.0—47.0
17	R	42.0—51.0
	L	45.0—47.0
18	R	37.0—38.0
	L	38.5—39.5
19	R	34.5—35.0
	L	33.0—35.0
20	R	0 —50.0
	L	0 —50.0
21	R	34.0—35.0
	L	34.0—37.0
22	R	46.0—48.0
	L	47.0—51.0
23	R	34.0—36.0
	L	34.0—37.0
24	R	27.0—42.0
	L	33.0—41.0

error that the differences in the hue discrimination between both eyes of all cases are pronounced around the middle of the spectrum where the limen is maximum.

It may be said that in other color types than deuteranomalia, the hue discrimination limen is much the same in both eyes. In deuteranomalia there seems to be a series of cases which have different hue discrimination limen between both eyes. The difference seems to be particularly definite around the maximum value of the limen. The number of examined cases in the present study is not still great enough. Therefore, a final conclusion should be drawn after an addition of experiments. However, the present conclusion seems to be endorsed by the fact that the all known unilateral (congenital) color anomaly is deutan.

It may be supposed also that the protan and deutan have different developmental mechanism. Its detail can not be explained at the present status of the knowledge. But this much may be supposed that the deutan may develop by an "intraocular" or "local" mechanism. This fact may also give an indication of the heredity of color defects.

Summary

This is a comparison of the hue discrimination between both eyes. The subjects are three normal cases, two cases of protanopia, four cases of deuteranopia, four cases of

protanomalia and eleven cases of deuteranomalia.

In deuteranomalia, the discrimination differs from one eye to another. The greatest difference is generally found around the maximum value of the hue discrimination curve (550 mμ).

This fact may be related to the previous experience that all unquestionable cases of congenital, monocular deficiency are deutan.

This fact also suggests that the developmental mechanisms of deficiency in deutan differs from those in the protan and that those in deutan is at least an "intraocular" or "local" defectiveness. It may also offer a problem to the heredity of color vision.

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OBJECTIVE MEASUREMENT OF OCULAR-AXIS BY X-RAY PHOTOGRAPH

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Introduction

Up to now the axial length was thought to be the most important as a responsible factor for refractive errors, especially myopia. The researches on the axial length of the living eye in the past, however, have used either calculation based upon measurements of the refractive power of the cornea and lens, or utilization of the light perecption caused by X-ray or the method of its determination of axial length. The former is indirect, the latter is subjective and that the both do not preclude a margin of error. In our laboratory a method which is conceivably more accurate has been studied, and Prof. Tutomu Sato and Dr. A. Nakajima reported a part of it.

The principle of our method is to measure the distance from the top of the cornea to the rear of the eye globe by X-ray photography making a layer of air around the globe. This being an objective method, the evaluation of the accuracy of the method can be obtained by animal experiment, and it has been able in such a way to prove the accuracy of our method. It has thus been able to test the accuracy of our method. By working on a photograph, we can eliminate for the most part, the effects of the subjective elements—on the part of both the patient and the measurer—involved in the previous methods.

Procedure

Air injection. Firstly, anesthesize the conjunctival sac by dropping 4% xylocain. Secondly, attach sclerocorneal contact lens on the eye globe. This lens prevents the air to come near the limbus. Thirdly, pull the upperlid upwards with a Desmarres retractor. Then the patient is asked to look down. Pick up bulbar conjunctiva with Tenon's capsule with an iris forceps at the point of about 4 mm. lateral from the lateral end of the insertion of the superior rectus, and pull up rather forcibly. Insert an injection needl at this point, and inject 5 cc of air. After the injection, remove the sclerocorneal contact lens. When experiment is finished the air is removed as completely as possible, with an injection needle.

Contact lens for taking photographs. The top of the cornea can not be demonstrated on the photograph by the condition which is suitable for the demonstrations of retrobulbar air layer. To avoid this dilemma, a corneal contact lens which contains tin powder was placed on the cornea. Thus, the lens was photographed, clearly, and the situation of the apex of the cornea was recognized. This lens is 0.5 mm. thick and 11.5 mm. in diameter. A set of 9 lenses with different radius of posterior curvature were prepared, and the lens which has nearest posterior curvature to that of the cornea was selected for each case. In such a case, the distance between the posterior surface of the lens and the anterior surface of the cornea is always less than 0.1 mm.. It was proved though our culculation that the maximal error of the axial length due to the difference of 0.1 mm. is less than 0.05 mm..

Photographing. Siemen's rotarics tube are used throughout, at the following condition. 64 KV, 100 MA. 1.2 second, distance between the tube and film : 100 cm..

The following two methods are used for the successful demonstration of the globe.

1st method ; Place the zygomatic bone and the body of the mandible of the patient on the cassette of the film. Measurer will adjust the patient's head position so as the sagittal suture will run at 30 degree to the film. (See illustration No. 1) Adjust the position of the tube so that X-ray which stands at right angle to the film passes through the rear pole of the globe. Ask the patient to look at an object so that the visual line of the eye to be measured will be parallel to the surface of the film. In this case the patient must hold the other eye fixed, because the eye to be measured is covered by an opaque lens. When the patient has squint, the object to be looked at will have to be adjusted according to the degree of his deviation.

Fig. 1 X-ray photographing

2nd method ; This differs with the first method only in the placing of the sagittal suture at 40 degree to the film.

Examination of the Method

Accuracy of the method. In our method, the X-ray tube is 1 meter from the film, and the axis of the eye to be measured is placed at a distance of 25 to 35 mm. from the surface of the film. The axial length appearing on the film must be found 2.5 to 3.5% longer than the actual figure.

This ratio of enlargement was examined rabbit eyes. After retrobulbar injection of air in rabbit eyes, their eye globe was photographed at a distance of 35 mm. from the film. The eye globe was then removed and measured directly with a micrometer. The enucleated globes were again photographed by X-ray at a distance of 35 mm. from the film. By comparing the three measurements, it was found that the ratio of 100 : 103.5 was exactly maintained.

The contour of the rear parts of the eye globes is slightly blurred due to the dispersion of the ray. But after some exercise in the measurement of axial length on photograph by use of an micrometer with its scale hidden, we could keep the margin of error under 0.2 mm..

Next, the axis of the globe should be found. This is easily known from the position of the image-making contact-lens. Since the lens almost covers the entire surface of the cornea, it shows the position of the cornea. A perpendicular line drawn from the center of the contact lens should be the ocular-axis. The lens may slide somewhat in the direction of a line perpendicular to the film due to gravity, but it does not slide in a plane parallel to the film.

Now, we must deal with the possibility of the additional inclusion of the tissues outside of the sclera in the apparent axial length. Judging from the fact that in photographs of both the human and the rabbit's eyes, the posterior parts of the globe is clearly shown, and there is little room to doubt that the rear surface of the sclera in the photograph is elongated by some retrobular tissues. The fact that the axial length of the living globe were identical in the experiments with rabbit globe seems to confirm this presumption.

The retrobulbar injection of Chinese ink was done on rabbits to confirm this point further. The rabbits were immediately killed and frozen with carbonic acid flakes. The skull, orbit, and eye globe were sawed together horizontally. Inspection of this showed that the injected matter spread directly behind the sclera. Also, after retrobulbar injection of air, the orbit of rabbits were opened from the orbit behind and made clear. We found that the Tenon's capsule was inflated by air. The eye globe with perfect Tenon's capsule were taken out, and was put in 4% formalin with a view to harden the tissues and cut along the axis. It was found that the air lay directly behind the globe. (See Illustration No. 2)

After these collations, it seems possible to conclude that our method has succeeded in measuring the axial length of the living eye.

Consideration on the complications. After the injection of air of 5 cc, not one case of

Fig. 2 Air in the Tenon's capsule.

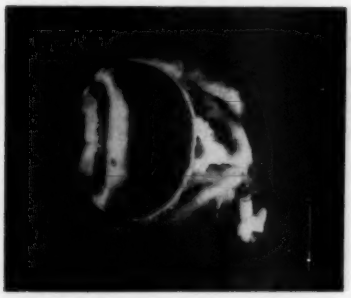


Fig. 3 X-ray picture of emmetropia.
(Table 1 Case 3)

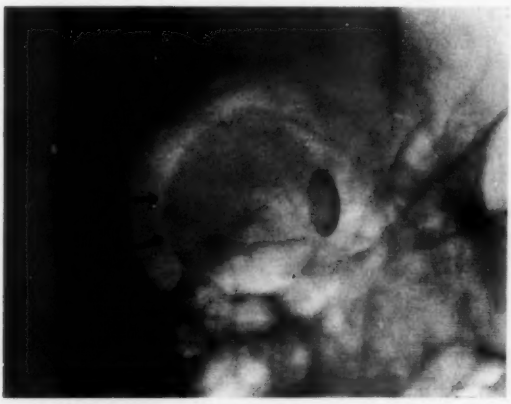
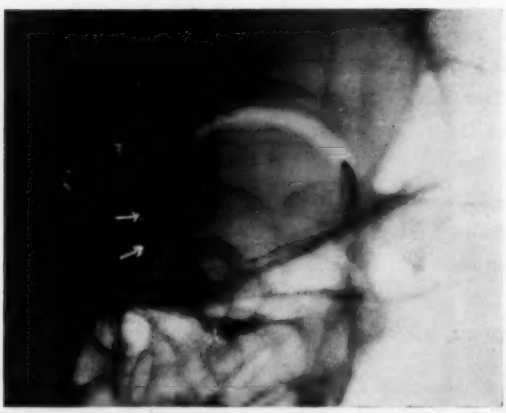


Fig. 4 X-ray picture of advanced myopia.
(Table 1 Case 13)



pain or discomfort has arisen. Subconjunctival bleeding was never seen.

The possibility of air embolus must be considered, since air is injected into the human body. But there is no large vessel near the bulbus, especially anterior to the equator. It will be easily seen by the measurer before injection if there should be an anomaly of vessel. Looking over previous reports of air injections into the human Tenon's capsule for other purposes, we could not find any cases where air embolism was reported to occur. Of course, it is advisable that the barrel of the syringe be pulled before the air injection, to ascertain that blood will not run. From these considerations, we feel our method is a safe one.

Imperfection of our method. That the thickness of the sclera and the choroid cannot be measured is a defect of our method. One mm. is decided to be subtracted from the measured reasult as their thickness. In spite of this defect, our method may be regarded as the only one method to obtain axial length of living eye with reliable accuracy.

Computation of axial length

3% is reduced from the "apparent" axial length which is obtained by the method described above. Then, 0.5 mm. for the thickness of the contact lens, and 1.0 mm. for the thickness of the sclera and choroid, are subtracted. The resulting figure is the optical axial length.

For instance, in case No. 3 in the table 1 the "apparent" axial length is 26.8 mm.. (Illustration No. 3) After a 3% reduction, and the subtraction of 1.5 mm.,

it gives 24.5 mm.. This is the real axial length of this emmetrope. In the case No. 13 (Illustration No. 4), "apparent" axial length is 33.4 mm. and the real axial length of this myope of -24D is 30.9 mm..

Results

The axial length of 14 eyes were measured by this procedure. The axial length of 3 emmetropic eyes, 3 myopias under -2.5D, 4 myopias of from -7 to -12D, and 4 myopias of from -20D is shown in the table 1.

Table 1. Results of Measurment

Case	Side	Subjectively refractive stage	Objectively refractive stage	Axial length on the film	Axial length
1	L	emmetropia	0	25.2	22.9
2	L	〃	0	26.1	23.8
3	R	〃	0	26.8	24.5
4	L	— 1.25	— 2.0	25.5	23.2
5	R	— 1.5	— 2.0	26.3	24.0
6	R	— 2.5	— 3.0	28.5	26.1
7	L	— 7.0	— 8.0	28.1	25.8
8	R	— 8.0	— 7.5	29.2	26.8
9	R	— 7.0	— 7.5	29.6	27.2
10	L	—12.0	—12.0	27.4	25.1
11	L	—20.0	—21.0	33.0	30.5
12	L	—24.0	—26.0	30.6	28.2
13	L	—24.0	—26.0	33.4	30.9
14	L	—24.0	—28.0	35.7	33.1

Discussion

The method of delineating the contours of the posterior parts of the globe by injection of air in Tenon's capsule was first experimented by Staunig and others on cows eyes. Since then, Gasteiger, Geisler, Spackmann, Hata, and others have used air injections in Tenon's capsule for localization of intraorbital foreign bodies.

Katz and others photographed cat eye globe with X-ray after injecting shadowing material in Tenon's capsule, and measured the axial length. They also injected showing material in Tenon's capsule of human eyes, but were unsuccessful because of resulting schrank eye and chronic inflammation. As far as we know, they have not published any data concerning to human axial length.

Witner, Droegenueller, Friedmann, Baltin, Dubilier, also took X-ray photographs after injecting air or shadowing material in Tenon's capsule, but none of them did so with the purpose of measuring axial length. In short, we have not heard of any reports concerning successful measurement of axial length of the living eye by objective method.

Our result so far obtained is too small in number to allow us to compare with the

results obtained by other methods in the past. We only describe of the method in this article.

Conclusion

We have succeeded in measuring the axial length of the living eye objectively by X-ray photographing. After animal experiments and various other considerations, it is concluded that this method is both accurate and safe. During our experiments, none of the patients whose eyes were measured suffered from harmful effects or pain. The axial length of 14 eyes incliding emmetropia, light myopia, and advanced myopia was measured by this method. The minimum was 22.9 mm. and maximum was 33.1 mm..

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DIFFERENTIAL DIAGNOSIS OF AMBLYOPIA

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Since the term amblyopia usually used is not a precise name of diagnosis but only a name of symptome such as poor visual acuity without marked organic changes, it may include various kinds of diseases—congenital amblyopia, ex anopsia, and other unknown diseases. While it is difficult to determine the accurate genesis of defective vision in each amblyopia, yet we have little knowledge of classification or differential diagnosis of amblyopia.

Materials

Eighty one cases of amblyopia form the basis of this study. The characteristics of them were as follows;

1. Visual acuity better than 6/12 was not obtained at the time of examination by correction glasses or other aids.

2. Pathologic changes were not usually present. When presented, they should not in themselves fully account for the subnormal vision.

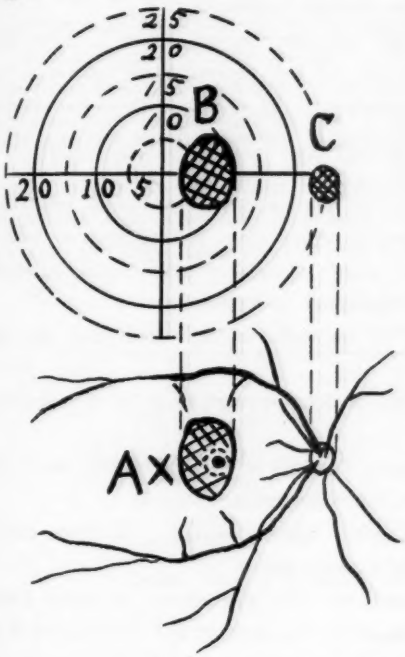
Among those, there were 51 cases of esotropia, 11 cases of exotropia, and 19 cases without squint.

Observations and Comments

Absolute scotomata in amblyopia

The unaffected eye was closed firmly and the affected eye was tested on perimeter and

campimeter whether it had absolute scotomata. In the cases of loose fixation, occlusion of the unaffected eye was attempted for several days until fixation became steady. Special care was taken, especially in the cases of eccentric fixation, to recognize the central scotomata which was not located in the center of the chart. It is easily demonstrated by observing the site of the blind spot, which is shifted from its normal position owing to eccentric fixation, whether the scotomata corresponds to the macular region of the retina. Figure 1 shows the relation between the eccentric fixation point, central scotomata and blind spot which were found in a case of divergent strabismus.



Fgi, 1. Absolute central scotomata in a case of eccentric fixation (right eye). A; eccentric fixation point. B; central scotomata. C; blind spot.

Absolute central scotomata or other type of absolute field-defect was investigated in 18 cases (25%) of all 60 patients (Table 1). These scotomatas did not subside after the occlusion of the unaffected eye for more than three days. Among these 18 cases, 13 cases had either pathological findings of the fundus or high refractive errors. Retinal corre-spondences were all normal in the cases combined with squint.

Although various types of field-defect in amblyopia have been reported by many authors, yet it has not been sure whether the scotomata was due to the suppression or congenital anomaly or an other disease. In those studies, the methods of examination were variable; some authors applied a binocular method such as red green test or mirror-screen test in which the unaffected eye fixed the test-object,

Table 1. Distribution of the Cases of Amblyopia according to the Findings of Visual Field

	number of cases			
	esotropia	exotropia	without squint	total
absolute central scotomata	6	1	4	11
another type of absolute scotomata	3	0	4	7
normal	42	10	11	63
total	51	11	19	81

while others employed an uniocular method in which the affected eye fixed the object under the condition of closing the other eye. In the former method, it is apt to demonstrate a facultative scotomata which differs considerably from an obligatory one. It is obvious that a facultative scotomata is caused by the inhibition which is excited in the squinting eye by the use of the macula of the other eye. Therefore, when one hopes to map out an obligatory scotomata, the uniocular method should be applied.

It is believed that if the suppression becomes deeper, a relative scotomata might change into an absolute. According to Chavasse, for example, obligatory scotomata may be found to be relative in least severe cases and to be absolute in less severe cases. There have been few experiments, however, to draw such conclusions and the following case suggests that even the most severe case does not retain the absolute scotomata.

Case 1. A boy Y., age 13, commenced his left divergent squint when 3 years old. The vision of the squinting left eye consisted only of the perception of light with the power of light projection. By occluding the unaffected eye, the visual aquity of the affected eye was restored quickly. Next day, when the visual field of the affected eye was examined, an absolute scotomata could never be demonstrated.

Although the relative scotomata, which is found in the squinting eye when tested by uniocular methods, may be dependent upon the effect of suppression, it is difficult to regard the absolute scotomata as the same. There are more reasons to believe that absolute scotomata is quite different from the suppression scotomata as follows;

1. The absolute scotomata was not subsided by occlusion of the unaffected eye for more than three days.

2. Any other portion of the retina except the region corresponding to the absolute scotomata was not influenced by suppression.

3. In most cases, there was found either organic changes or high refractive errors in the affected eyes which seemed to be the cause of the scotomatas.

4. Absolute scotomata was demonstrated in 8 cases without squint. In these cases, the suppression should never take place under any circumstances.

Moreover it is noticeable that, in the cases combined with strabismus, no cause could be found of the squint, except ths visual disturbance of the affected eye. Therefore it is suggested that the absolute scotomata has no relation to the functional adaptation such as the inhibition and that the group of amblyopia with absolute scotomate differs essentially from the group without it and strabismus may occur in some cases owing to the defective vision which affects the fusion faculty.

Some authors have considered that absolute scotomata is due to a hemorrhage that occured in the retina in the macular region during birth but has no ophthalmoscopically visible traces. Absolute scotomata may also be found as a symptome of a certain congenital anomaly or some disease such as retrobulbar neuritis. Although the cause of the scotomata is not determined always in every case, it is sure that we must segregate these cases from the cases of amblyopia ex anopsia and we may call them pathologic amblyopia.

Variation of the fixation in amclsopia

Exclusive of 18 cases with absolute scotomata, 63 cases of amblyopia were examined whether they fixated the test object with the fovea or another peripheral part of the retina. Eccentric fixation was demonstrated easily by observing the dislocation of the blind spot, but more accurately by observing the fundus with the Zeiss slit lamp.

Central fixation was demonstrated in 11 cases (17%), among which 4 cases had strabismus with normal correspondence and other 7 cases had no deviations. Every one of them had either some organic change such as blurring of the edges of the disc, deviation of the macula, anomaly of the orbita, etc., or high refractive error such as high hypermetropia, high myopia or anisometropia. On the contrary, such pathological findings were rather rare in the group without central fixation (Table 2).

In the eccentric fixation group, including the cases of lost fixation, 42 cases (80%) were found to be associated with convergent or divergent strabismus, among which anomalous correspondence was demonstrated in 18 cases. These findings are summarized in Table 3.

It has been considered that fixation is influenced by the depth of suppression; central fixation is maintained in mild cases of amblyopia, while if the suppression is persistently and more deeply present, fixation would become eccentric. It is difficult, however, to admit such consideration to be true because of following reasons. From the

Table 2. Organic Changes or High Refractive Errors demonstrated in the Cases of Central and Eccentric Fixation (exclusive of 18 cases with absolute scotomata)

	central fixation	eccentric fixation
anomaly of the disc	4	5
anomaly of the retina	2	1
anomaly of other portions	1	1
high refractive error	4	1
without marked changes	0	44
total	11	52

Table 3. Distribution of Cases of Amblyopia according to the Condition of Fixation. a.c.; Anomalous Correspondence. n.c.; Normal Correspondence

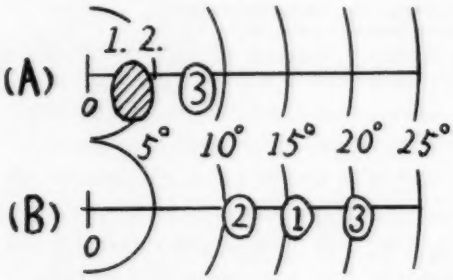
fixation		esotropia	exotropia	without squint	total
central	a.c.	0	0	0	0
	n.c.	2	2	7	11
eccentric	a.c.	14	4	0	18
	n.c.	26	4	4	34
total		42	10	11	63

observations, summarized in Table 2 and 3, there were found marked differences between the central and eccentric fixation group. In the former group, strabismus was rare and all had some organic changes or high refractive errors which might be the cause of the defective vision although they could not in themselves fully account for amblyopia. Correspondence was normal in all cases associated with squint. On the contrary, those with eccentric fixation were combined with strabismus as a rule and anomalous correspondences were found in many cases. Moreover most cases had neither organic changes nor high refractive errors.

Therefore, in the former group, amblyopia is mainly due to either an organic change or a high refractive error and has little relation to the suppression. Strabismus is not always an indispensable condition, while it may occur in some cases owing to the defective vision. This group should never be called suppression amblyopia but pathological amblyopia.

The latter group seems to be closely connected with strabismus and hardly any cause of defective vision may be demonstrated in most cases. These amblyopes with eccentric fixation should be called amblyopia ex anopsia or suppression amblyopia because the cause ot amblyopia may never be found except upon the functional disturbance. Visual acuity is less than 1/6 in these cases.

Fig. 2. Dislocation of the blind spot in various conditions in a case of eccentric fixation with right esotropia. Amblyopic right eye fixes uniocularly the test object which is placed in the front (1), in the right side of 30°(2), in the left side of 30°(3), before operation (A), after the squint operation (B).



Although it is believed that eccentric fixation may be the manifest result produced by the suppression upon the macular region, yet there remains some doubt upon the matter. Recently Böhme has pointed out that the eccentric fixation point is not an immobile one as ever thought but fairly displaceable according to the horizontal movement of the gaze and may become almost central in the primarly position after a certain operation of horizontal muscles which effects the marked improvement of the visual acuity. Figure 2 shows the same phenomenon as Böhme has reported but it is demonstrated more clearly with the change of the site of the blind spot. It is evident that the fixation point is removed towards the macular region in the primary position after the squint operation.

If the development of the eccentric fixation is due to the defective vision of the central field according to the suppression in the macular region of the retina, the fixation point should never become central under any circumstances and visual acuity also should never be improved. The fact suggests, that the main cause of eccentric fixation is not the suppression but the weakness of the faculty to maintain the central fixation which should

be regarded as an attribute of amblyopia ex anopsia itself.

Summary

For the purpose of differentiating pathological amblyopia from amblyopia ex anopsia, the authors examined 81 cases of amblyopia and obtained the following results.

1. The cases in which absolute scotomata will appear in the amblyopic eye unilaterally are due to the disturbance of vision caused not by the suppression but by the eye disease that brings about scotomata.

2. The cases of complete central fixation of the amblyopic eye are mainly due to disturbance of vision caused by organic disease or high refractive error.

Above two groups should be called pathologic amblyopia and segregated strictly from the following group.

3. The cases to be called amblyopia ex anopsia are unable to fix the eye in the central portion and do not prove the scotomata, and rarely have organic diseases or refractive errors of the eye. These cases are responsible for the disturbance of vision resulting in strabismus and visual acuity is less than 1/6.

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THE STUDY OF THE EFFECT OF AMINO ACIDS ON THE REGENERATION OF THE VISUAL PURPLE (PART I)

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Introduction

The regeneration of the visual purple plays the important role for the function of rods of the retina in the dark. There are many factors which influence on the regeneration of the visual purple. For instance hypophysis, hypothalamus, liver, drugs and temperature.

The visual purple consists of carotenoid and proteine so called "opsine".

It has been recognized that opsine contains many amino acids.

Then, the effect of amino acids must be one of many factors which influence the visual purple regeneration.

But in literature about the effect of amino acids on the visual purple regeneration has not been reported satisfactorily.

In this paper, the effect of amino acids on the visual purple regeneration is reported and the mechanism is discussed.

Experimental method

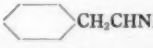
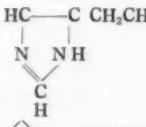
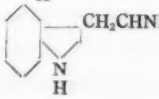
Test animals. adult bull frogs (Rana catesbiana Shaw) were used.

Period of experiment. from June 1956 to October 1957.

Temperature of experiment in dark room. kept 18°C by air conditioning.

Extraction and measurement of visual purple. Outer segments were separated from the retina by Prof. Kimura's method[1]. Visual purple was extracted by 1% digitonine

Table 1. Visual Purple Densities

Amino acids		Duration of period of dark adaptation in minutes		
		30	60	120
1 aqua dest. (contrast)	H_2O	0.108	0.155	0.220
2 Glycine	CH_2NH_2COOH	0.150	0.177	0.264
3 Alanine	$CH_3CH\ NH_2COOH$	0.276	0.268	0.285
4 Serine	$CH_2OH\ CHNH_2COOH$	0.155	0.142	0.300
5 Valine	$\begin{matrix}CH_3\\CH_3\end{matrix}\!\!>\!CH\ CHNH_2COOH$	0.260	0.310	0 310
6 Isoleucine	$\begin{matrix}CH_3\\C_2H_5\end{matrix}\!\!>\!CH\ CHNH_2COOH$	0.285	0.310	0.268
7 Glutamic acid	$COOH\ (CH_2)_2\ CHNH_2COOH$	0.150	0.168	0.230
8 Leucine	$\begin{matrix}CH_3\\CH_3\end{matrix}\!\!>\!CH\ CH_2CHNH_2COOH$	0.173	0.220	0.300
9 Arginine	$\begin{matrix}NH\\NH_2\end{matrix}\!\!>\!CNH\ (CH_2)_3\ CHNH_2COOH$	0.138	0.251	0.280
10 Ornithine	$H_2N\ (CH_2)_3\ CHNH_2COOH$	0.170	0.238	0.339
11 Phenylalanine		0.318	0.278	0.329
12 Tyrosine		0.261	0.324	0.263
13 Histidine		0.184	0.206	0.229
14 Tryptophane		0.204	0.238	0.268

solution placing it during 24 hours in an ice box at 4°C.

The value of the regeneration of visual purple (A) was expressed as follows:

the absorption coefficient was measured at 500 mμ by photoelectric spectrophotometer.

$A = \frac{E_1 - {}_0E}{W}$ E_1: before bleaching

E_0: after bleaching

W: weight of dried outer segments.

Procedure of experiment: each amino acid was prepared as 3/100 M aqueous solution. Aque dest. was used as control.

Frogs were injected with the amino acid solution immediately, after they were adapted to light (6000 Lux) for 20 minutes and next were adapted to the dark for 30, 60 and 120 minutes.

One group is consisting of 5 frogs (10 eyes).

Results

The results were shown in Table 1.

1) Phenylalanine, tyrosine, tryptophane, alanine, valine and isoleucine augmented remarkably the regeneration of visual purple.

2) Leucine and ornithine augmented considerably the regeneration of visual purple.

3) Histidine, arginine, serine, glycine and glutamic acid augmented slightly the regeneration of visual purple.

Discussion

We have many theories about the combining of the carotenoid with proteine.

Dr. Morton[2)3)] reported the theory of N-bond. Dr. Morton and his cooperators made out a substance alike indicator yellow by the conbination of retinene with amines. This combination occurred in an alkaline medium and these λ max was at 440–450 mμ, therefore it resembled the visual purple. While it had been recognized that the visual purple was regenerated most remarkably in alkaline medium.

Dr. Wald[4)5)] reported the theory of S-bond. As the reason, he asserted, that the substances possessing the SH-group augmented the regeneration of visual purple and the SH-group was isolated by bleaching of the visual purple.

Prof. Hosoya[6)] reported that the substances possessing an unstable CH_3-group augmented the regeneration of visual purple. And so he maintained that the unstable CH_3-group played an important role in the visual cycle.

Dr. Hayano[7)] reported that transaminase existed in the retina and influenced augmentatively the visual purple regeneration.

When these theories are compared with our results, it is supposed that the amino group plays an important role for the regeneration of visual purple in the living body of frogs. Judging from our results and these theories, the following matters may be considered

for the mechanism of the augmentative effect of amino acids on the visual purple regeneration.

1) In the absorption spectrum of the visual purple solution, the absorption of proteine (γ zone) exsists at 278 mμ. This fact shows, that the proteine of visual purple includes aromatic amino acids. Besides, it is known that glutamic acid, alanine and aspartic acid etc. are found in visual purple solution by the method of paperchromatography and paper-electrophoresis.

2) The retinene which is aldehyde of vitamine A, has a quality to combine with the amino group contained in amino acid, primary amine and peptide in alkaline medium. While, it is known that the visual purple is regenerated most remarkably in alkaline medium.

As the λ max of this combined substance is at 440–450 mμ (near the λ max of visual purple), the possibility of this combination is very large.

3) Retinene combines with the amino group by the above related reason, but when the substance made by combination is not a physical one, sometimes it inhibits the visual purple regeneration for example in hydroxylamine and dihydroxyphenylalanine.

They combine with retinene by their amino group, but the physical regeneration of visual purple may be inhibited in the struggle for combination of the retinene with the amino group, as the products are not physical substances.

By the above related mechanism, most of the amino acids which have been experimented with by us, shall have an augmentative effect on the regeneration of visual purple.

In conclusion, it may be said that these amino acids augment the regeneration of visual purple, as they shall exist in the components of visual purple and shall play a role for the combination with the retinene.

The difficulty of the pure separation of visual purple proteine has been keeping the research of opsine in a backward condition comparing it with the research of its carotenoid prosthestic group. As the visual purple extracted with digitonine solution is combined with digitonine molecules, it becomes a big molecule and it is very difficult to separate the visual purple molecule alone.

Accordingly, in the present step we have not yet obtained a satisfactory understanding about the mechanism of the augmentative effect of amino acids on the visual purple regeneration. But we believe that much important knowledge about the mechanism of the visual purple regeneration is obtained in this study.

Summary

The effect of various amino acids on the regeneration of visual purple was examined. Fresh bull frogs were used as the test animals. The concentration of visual purple of the premedicated and the control animals were measured by photoelectric spectrophotometer.

In the results it was shown that:

1) phenylalanine, tyrosine, tryptophane, alanine, valine and isoleucine augmented remarkably the regeneration of visual purple,

2) leucine and ornithine augmented considerably the regeneration of visual purple,

3) histidine, arginine, serine, glycine and glutamic acid augmented slightly the regeneration of visual purple.

The mechanism was discussed.

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THE CHANGES OF THE RETINAL CIRCULATION AS A SEQUELA OF THE LATE TOXEMIA OF PREGNANCY

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Introduction

The symptoms of the toxemia of pregnancy have been regarded to be characterized by the spastic contraction of the minute vessels and capillaries. The retinal vessels also were found, by the ophthalmoscopic examination, to exhibit a spastic contraction. However, little has been known about the development of this vascular change of the retina after the labor.

In recent years, Uemura and Kawashima[4] reported a new electronic sphygmotonometer, by means of which the corneal pulse-waves can be recorded automatically. The corneal pulse-waves are believed to result chiefly from the pulsation of the retinal artery and to represent the state of retinal circulation. Hence the method to analyze the corneal pulse-waves, according to the theories of circulatory dynamics, was put forward by these authors; and it has become possible to detect changes of the retinal circulation which can hardly be recognized by the ophthalmoscopic examination.

The present investigation was, therefore, undertaken to know how long and to what extent the changes of the retinal circulation due to the toxemia of pregnancy persist after the labor. By means of the electronic sphygmotonometer, the corneal pulse-waves were

recorded from women having anamnesis of the labor with late toxemia of pregnancy. By an analysis of these corneal pulse-waves, it was found, that remarkable changes of the retinal circulation were retained even 4 years after the labor, although the ophthalmoscopic fundus changes were improved to some extent with the passage of time.

Materials and Methods

One hundred and forty women, having anamnesis of labor with the so-called mild type of preeclampsia (expressed briefly as the pathological labor in the following description), were examined; the age of the patients ranged from 20 up to 47 years. Forty one women, from 21 up to 37 years old, with anamnesis of normal labor were also examined as a control. The patients were divided, according to the intervals after the labor, into 6 groups, i.e. groups of 0.5, 1, 2, 3, 4 and 5 years.

Besides the examination on the general state of patients, routine measurements of the blood pressure were carried out, and the ocular tension was measured with Schiötz's tonometer. Ophthalmoscopic examination was also performed, and the changes of the fundus were graded according to the classification of Wagener, Clay and Gipner[5].

The corneal pulse-waves were recorded by means of "The Recording Electronic Sphygmotonometer" (Uemura and Kawashima), and the blood pressure of the retinal artery (R.A.P. max. and min.) was obtained therefrom. The extent of the area defined by the corneal pulse-wave and the base line (briefly the area of pulse-wave) was measured and expressed in square mm.. At the same time, E.C.G. was recorded by the 2. lead, in order to know the intervals of individual pulses and the duration of diastole.

In the circulatory dynamics, several coefficients have been used to represent the state of blood flow and the characteristics of the wall of vessels, e.g. the volume coefficient, the resistance of the peripheral blood flow, the stroke volume, and the volume of arterial cavity at the end of systole etc.. It is believed that these concepts can be expanded to the retinal circulation. Hence the equivalents of these coefficients, which represent the state of the retinal circulation, can be obtained, using the values of R.A.P. and of the area of pulse-waves; the calculation of these coefficients is called as the analysis of the corneal pulse-waves. In the present investigation, the following coefficients were calculated.

1. *C-R (Clinical Rigidity)*. Kawashima[2] and Yamamoto[7] introduced the concept of clinical rigidity, which represents the resistance of the wall of vessels against the pressure applied from outside. The clinical rigidity was computed from the following equation:

$$\text{C-R}=\frac{\text{Area of pulse-wave under definite load (10 and 20 mmHg)}}{\text{Area of pulse-wave at diastolic pressure of the retinal artery}}$$

The increase of C-R indicates the increased rigidity of the wall of vessels.

2. *E-Retina (The volume elasticity coefficient of the retinal artery)*. The volume elasticity coefficient of the retinal artery was, as calculated by Yamamoto, given by

$$\text{Log.}\frac{\text{The diastolic pressure of the retinal artery}}{\text{Area of the pulse-wave at the diastolic pressure of the retinal artery}}$$

The increase of the E-Retina is regarded to be proportional to the decrease of the elasticity of the wall of vessels.

3. *rs/rd-Carotis* (*The volume elasticity coefficient of the carotid artery*). The volume elasticity coefficient was calculated also for the carotid artery. This coefficient was derived from

$$\text{rs/rd-Carotis} = \sqrt[4]{\frac{(\text{R.A.P. max.}-T_0)\ (\text{Br. A.P. min.}-\text{R.A.P. min.})}{(\text{R.A.P. min.}-T_0)\ (\text{Br. A.P. max.}-\text{P.A.P. max.})}}$$

(by Weigelin[6]),

where rs means the radius of the carotid artery when the heart is in its systolic phase, rd means the radius of the carotid artery in a diastolic phase, Br. A.P. is blood pressure measured at the brachial artery, and T_0 is the ocular tension respectively. The decrease of rs/rd-Carotis means the decrease of the elasticity of the wall of the carotid artery.

4. *W-Retina* (*Resistance of the peripheral blood flow of the retina*). By analogy with Broemser, Ranke and Laubers' formula for the peripheral blood flow, the W-Retina was calculated from

$$\frac{\text{R.A.P. min.} \times \text{E-Retina} \times \text{D}}{\Delta P}$$

where Δp means the difference between the systolic and diastolic blood pressure of the retinal artery, and D is the duration of diastole respectively. The increase of the resistance of peripheral blood flow indicates the increased spastic tendency and/or advanced sclerotic change of the wall of vessels.

5. *i-Retina* (*The velocity of retinal blood flow*). The velocity of blood flow was, by a modification of Wezler and Bögers'[1] formula, derived from the following equation:

$$\text{i-Retina} = \text{Pm/W-Retina}.$$

Where Pm is given by R.A.P. min. $+ \Delta P/3$, which is equivalent to the mean value of retinal blood pressure. It is believed that the raised Pm indicates the increase of spastic tendency of minute vessels.

6. *Vsp-Retina* (*The volume of the cavity of the retinal artery at the end of the systole*) *and Vs-Retina* (*The stroke volume of the retinal artery*).

Vsp-Retina could be computed, by modifying the formula of Broemser, Ranke and Wetterer[1], from

$$\Delta P/\text{E-Retina}$$

On the other hand, the stroke volume Vs is given, as shown by Broemser and Ranke, by $T/D \times Vsp \times Z$ for blood vessels. By analogy with this view, the Vs-Retina was obtained by substituting Vsp for Vsp-Retina. In this formula, T indicates the intervals of individual pulses, D is the duration of a diastole and Z is the correcting coefficient which is equal to 0.48. The decrease of Vs-Retina and Vsp-Retina indicates the decrease of the volume of blood flow.

Results

Fundus changes. Fundus changes, observed in 6 groups of women with anamnesis of

Table 1. Fundus Changes

Fundus Changes		Controls		Patients	
		Number of Cases	Per cent	Number of Cases	Per cent
Changes of Artery	Narrowing of Arterial Column	5	12.2	75	53.57
	Copper-wire Artery	21	51.22	95	67.85
	Silver-Wire Artery	0		1	0.71
	Sheathing of Artery	0		22	15.71
	Irregularity of Caliber	0		2	1.43
Changes of Vein	Tortuosity	9	21.95	33	23.57
	Congestion	19	46.34	106	75.72
	Arterio-venous Crossing	7	17.07	40	28.57
Other Changes	Guist's Phenomenon	0		1	0.71
	Peripapillar Opacity	0		40	28.57
	Number of Cases Examined	41		140	

Table 2

Correlation between the Degree of Fundus Changes and the Intervals after the Labor

Degree of Fundus Changes \ Intervals (years)	0.5	1.0	1.5	2.0	2.5	3.0	3.5	4.0	4.5	Total
0	1	2		6	3	4	2	2	1	21
Ⅰ	4	2	4	2	4		2	4	1	23
Ⅱ	11	7	12	4	4	8	4	5	1	56
Ⅲ	11	11	4	1	1	5		6	1	40
Total	27	22	20	13	12	17	8	17	4	140

Coefficient of Correlation: $r = -0.2088$, Significant.

$F_0 = 6.2911$, $n_1 = 1$, $n_2 = 138$, $3.92 > F > 3.91$, $\therefore F_0 > F$, Significant.

Ratio of Correlation: $\eta^2 = +0.0439$, Insignificant.

$F_0 = 2.0815$, $n_1 = 3$, $n_2 = 136$, $2.68 > F > 2.67$, $\therefore F_0 < F$, Insignificant.

the pathological labor, are summarized in table 1. In this table, fundus changes observed in women who passed normal labor, are also shown as a control. As readily seen in the table, a considerable number of patients had vascular changes in the fundus compared with the contral.

In the present investigation, it was of particular interest to know, how the fundus

changes developed with the passage of time after the labor. It was, therefore, examined whether there was a correlation between the intervals after the labor and the degree of fundus changes. Then, a significant negative correlation was obtained between them as shown in table 2. This result indicates that the fundus changes subsequent to the toxemia of pregnancy were improved to some extent with the passage of time after the labor.

Analysis of corneal pulse-waves. The corneal pulse-waves were analyzed according to the method mentioned above and R.A.P. min., R.A.P. max., C-R (10 mmHg and 20mmHg), E-Retina, W-Retina, Vs-Retina, Vsp-Retina, i-Retina, Pm and rs/rd-Carotis were calculated. Then it was statistically examined whether the values obtained from patients showed some differences from the control. The results in 6 groups patients are given in table 3.

As seen in the table, maximum blood pressure was slightly raised only in groups of 2 and 3 years intervals. On the other hand, a significant elevation was found in R.A.P. min. in 5 groups except the group of intervals of 5 years. R.A.P. max. was also considerably raised in groups of 0.5, 3 and 4 years intervals. A significant elevation was also found in Pm in groups of 0.5 and 4 years.

Table 3. Analysis of Corneal Pulse-Waves

	Controls	Patients											
Intervals (years)		0.5		1		2		3		4		5	
Number of Cases	41	27		22		33		29		25		4	
R.A.P. min.	35.220 ±4.967	39.870 ±3.829	+	39.545 ±4.950	+	40.606 ±6.799	+	44.103 ±5.225	+	40.420 ±3.597	+	36.125 ±4.625	
R.A.P. max.	69.488 ±6.134	77.463 ±7.890	+	70.295 ±7.686	−	71.818 ±0.601	−	74.914 ±7.566	+	77.680 ±7.373	+	76.125 ±9.563	
C-R 10 mmHg	0.605 ±0.185	0.706 ±0.175	−	0.691 ±0.152	−	0.600 ±0.167	−	0.659 ±0.176	−	0.759 ±0.140	+	0.536 ±0.070	
C-R 20 mmHg	0.445 ±0.151	0.527 ±0.177	−	0.496 ±0.135	−	0.446 ±0.170	−	0.431 ±0.170	−	0.517 ±0.158	−	0.368 ±0.066	
E-Retina	1.394 ±0.177	1.627 ±0.134	+	1.595 ±0.159	+	1.618 ±0.213	+	1.607 ±0.160	+	1.608 ±0.202	+	1.156 ±0.104	
W-Retina	0.446 ±0.166	0.618 ±0.231	+	0.667 ±0.255	+	0.623 ±0.347	−	0.714 ±0.288	−	0.624 ±0.266	+	0.494 ±0.185	
rs/rd-Carotis	1.206 +0.161	1.178 ±0.163	−	1.114 ±0.106	−	1.169 ±0.155	−	1.187 ±0.103	−	1.232 ±0.150	−	1.241 ±0.134	
Vsp-Retina	24.851 ±5.301	23.577 ±6.629	−	19.602 ±4.975	+	21.250 ±5.542	−	21.413 ±5.158	−	24.079 ±5.979	−	35.307 ±9.299	
Vs-Retina	33.279 ±7.444	30.008 ±8.416	−	27.967 ±9.529	−	34.887 ±12.090	−	28.712 ±9.826	−	33.205 ±9.264	−	34.683 ±8.143	
Pm	47.000 ±4.943	52.401 ±3.129	+	49 795 ±5.011	−	47.737 ±6.670	−	50.986 ±4.042	−	52.597 ±4.020	+	49.958 ±5.958	
Br. A.P. min.	67.366 ±10.167	72.519 ±12.022	−	66.545 ±9.091	−	68.875 ±13.047	−	72.786 ±10.505	−	70.364 ±10.273	−	63.000 ±5.000	
Br. A.P. max.	117.829 ±11.125	127.741 ±15.268	−	121.364 ±12.157	−	127.313 ±15.934	+	127.821 ±15.952	+	120.409 ±16.719	−	119.500 ±11.500	

\+ Significant
− Insignificant

Increased E-Retina was obtained in groups of 0.5, 1, 2, 3 and 4 years intervals. A remarkable augmentation of W-Retina was also found in groups of 0.5, 1, 3 and 4 years. The clinical rigidity (10 mmHg), however, showed an increase only in a group of 4 years intervals. On the contrary, a decrease was found in Vsp-Retina of the 1-year-group.

These results indicate, that remarkable changes of the retinal circulation and the wall of vessels, similar to that in hypertensive diseases, were caused by the late toxemia of pregnancy. Moreover, no significant correlation was found between R.A.P. and the blood pressure. Judging from these facts, it is reasonable to conclude that a tendency of intracranial hypertension persisted, due to the toxemia of pregnancy, even at intervals of several years after the labor.

Besides the statistical studies mentioned above, studies were also made on the correlation of the analytical values with the intervals after the labor and the degree of fundus changes. However, no significant correlation was proved.

Influence of repeated labors. In order to know the influence of repeated, normal and pathological labors, the following studies were performed.

The patients were divided into 3 groups; 1. patients who repeated 2 or more pathological labors, 2. patients who passed normal labors previous to the pathological one and 3. patients who passed only one pathological labor. The last group served as a control.

In the group of patients with previous normal labors, slight differences were found in the Vs-Retina and C-R (10 mmHg) compared with control. On the other hand, in the group of patients with repeated pathological labors, differences from the control were found in many more coefficients, i.e. Vsp-Retina, Vs-Retina, W-Retina, i-Retina, and in maximum and minimum blood pressure.

Judging from these results, the following may be concluded: repeated pathological labors caused more severe changes in the retinal circulation; and even normal labors, when repeated, could advance the circulatory changes of the retina.

Discussion

The fundus changes due to the toxemia of pregnancy was, by the ophthalmoscopic examination, thoroughly studied by Tano[3]. He could not, however, make any investigation on the development of these fundus changes after the labor. In the present study, women having anamnesis of labor with the so-called mild type of preeclampsia were examined, and a considerable number of patients had fundus changes, situated chiefly on the retinal vessels, several years after the labor. These changes, however, underwent an improvement with the passage of time.

There have been no analytical studies of the corneal pulse-waves for women who passed pathological labor. In the present study, it was proved, by the analysis of the corneal pulse-waves, that remarkable changes of the retinal circulation, which could hardly be recognized by the ophthalmoscopic examination, were retained even 4 years after the

labor. These changes of the retinal circulation could be considered to be the same in nature as the vascular changes which occur in hypertensive diseases. In addition, the blood pressure of retinal artery was significantly raised compared with the control, although the blood pressure revealed only a slight elevation. It may, therefore, be concluded that a tendency of intracranial hypertension persisted, due to the toxemia of pregnancy, even at intervals of several years after the labor. Moreover, no significant correlation between the blood pressure and the blood pressure of retinal artery was found. This fact may also support the conclusion mentioned above.

Not only pathological labors, but also even normal labors, when repeated, cause more severe changes in the retinal circulation. This result is not far from the assumption that toxemia of pregnancy can play some role in the manifestation of hypertensive diseases of women. In the present state of our knowledge, however, there is no decisive evidence supporting this assumption; hence it requires further ellucidation to solve this problem.

Summary

The retinal circulation of patients, who passed the labor with toxemia of pregnancy, has been studied by means of the electronic sphygmotonometer, with following results.

1. The fundus changes due to the so-called mild type of preeclampsia were found to be situated chiefly on the retinal vessels.

These fundus changes could be improved to some extent with the passage of time after the labor.

2. The analytical study of the corneal pulse-waves pointed out that there was a significant difference in the retinal circulation between the patients with normal labor and with pathological one, even at intervals of 4 years after the labor. These changes of retinal circulation were proved to be the same in nature as that observed in hypertensive disease.

3. The blood pressure of the retinal artery was, in patients with pathological labor, at a cigher level than normal. On the other hand, the blood pressure remained in the normal range, and no significant correlation between the blood pressure and the blood pressure of the retinal artery was found. From these facts, it was concluded that a tendency of intracranial hypertension was retained after the pathological labor.

4. Not only pathological labors, but also even normal labors, when repeated, caused an advance of the pathological changes of the retinal circulation.

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EXPERIMENTAL STUDIES ON THE OCULO-MOTOR CENTERS

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Introduction

Although nuclei in the brain stem have been considered as the intermedial center of the ocular movements, few reports are found on the relation between diencephalon and the ocular movements.

Spiegel and Scala confirmed that conjugate downward movements could be provoked by the electrical stimulation of the ventral portion of the grey matter under or just at the level of posterior commissure and conjugate upward movements by the stimulation of the posterior portion of the thalamus or the midportion of the tegmentum of the anterior colliculus.

Rioch and Brenner reported, that in the decorticated cats, the electrical stimulation of the head of the caudate nuclei (caput nuclei caudate) resulted in the contraversive movements of both the head and the eye.

Spiegel and Scala, commenting on the destruction experiment of the superior colliculi carried out by Bechterew, stated that it might be due to the destruction of the mesencephalic aquaeductus or posterior commissure nuclei.

Though the general ideas of oculo-motor centers have been established, the author added some experimental results on the electrical stimulation of the various parts of diencephalon in cats, expecting that the diencephalon has some intimate relations with the ocular movements.

Experimental Method

Thirty-four adult cats of about 2kg body weight were used as expeaimental animals.

Electrical stimulation was applied through bipolar electrodes with an interelectrodal distance of 1.2 mm.. The output of the electro-stimulator was isolated.

The cat was anesthetized with chloroform and its extremities were fixed and the head was lightly fixed with supporting the neck and the external acoustic meatus. The skin of the head was cut 30 mm. in sagittal direction and a small hole of 1.5 mm. diameter was made on the cranium and the electrode holder (Kurotsu type) was fixed there.

Two hours after operation, the stmulation experiments were begun. Square wave of 1-5 volts was used. The wave form was checked on the cathode ray oscilloscope. After the experiments, the brain tissue was fixed with 10% formalin, embedded in celloidin, cut in frontal serial section of 15μ thick, staind with haematoxylin-eosin and when necessary, with Nissl's or Yashiro's method. Winkler and Potter's nomenclature was used for the topographical describing.

Experimental Results

The exact ocular movements were observed in fifteen cats.

These results were shown in Table 1.

Table 1

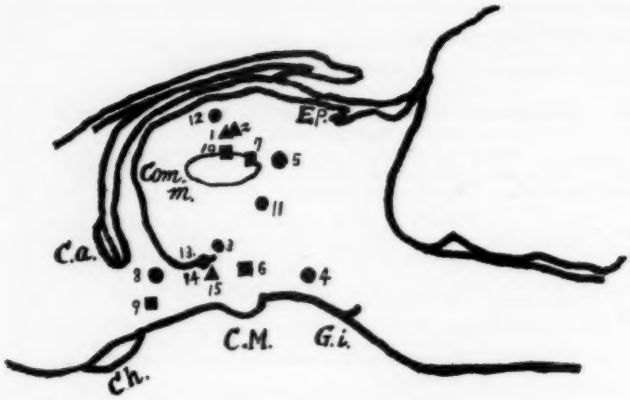
Case	Stimulus	Ocular movement			Position of the electrode tip (Histrological observation)
		Eye-ball	Direction	Kind	
No. 1	2~4 V. c/s.	Binocular	Homo-lateral	Conjugate deviation	Lateral part of habenula and upward part of medial thalamic nuclei b.
No. 2	2~4	Binocular	Homo-lateral	Conjugate deviation	about same part of No. 1. but merely backward of it.
No. 3	2~4	Monocular	Contra-lateral	horizontal	Medial part of columna fornicis and the place where the anterior infundibulum shifts to posterior.
No. 4	1~3	Monocular (Binocular)	Contra-lateral	horizontal	Oculo-motor nerve root.
No. 5	2~5	Monocular (Binocular)	Contra-lateral	horizontal	From the upper end of the medial thalamic nuculeus to the lower end of the Meynert's fasciculus.
No. 6	4	Monocular	Upward	vertical	Medial part of lateral hypothalamic nucleus and interior part of medial hypothalamic nucleus between the lateral hypothalamic nucleus.
No. 7	3~5	Monocular	Upward	vertical	Lateral part of medial thalamic nuclei b and the upperward part of Meynert's fasciculus.
No. 8	6	Monocular (Binocular)	Contra-lateral	horizontal	Latero-posterior part of chiasma and lateral hypothalamic nucleus around the end of the internal capsule.
No. 9	6~8	Binocular	Upward	vertical	caudal part of the medial infundibulum shift.
No. 10	2~5	Binocular	Upward	vertical	Lateral part of medial thalamic nuclei b and the peripheral part from the center.
No. 11	3	Binocular	Contra-lateral	horizontal	Inferior part of the lateral hypothalamic nucleus and mid-portion between nucleus proprius of the cerebral peduncle and lateral hypothalamic nucleus.
No. 12	4~8	Binocular	Upward	vertical	pulvinar at the peripheral part of the anterior thalamic nucleus.
No. 13	4~6	Binocular	Contra-lateral	horizontal	Nucleus proprius at the cerebral peduncle at the latero-posterior part of the lateral hypothalamic nucleus.
No. 14	4~6	Binocular	Contra-lateral	horizontal	Inferior part of nucleus proprius of the lateral cerebral peduncle at the posterior part of the lateral hypothalamic nucleus.
No. 15	4~6	Binocular	Contra-lateral	Conjugate deviation	antero-lateral part of mamillary body

Comments and Summary

Even though the general idea of the central of ocular movements has been established by many animal experiments, study of autopsy cases and clinical observations, many anatomical and physiological problems remained unsolved. It is obvious that the cortico-medullar tract is concerned with the ocular movements. However, there are many uncertain points on this tract and almost nothing was found on the pyramidal tract which is concerned with ocular movements.

There are some people who consider that efferent impulses arising in the cortical center are directly transmitted to the peripheral nerve nuclei and result in ocular movements.

Fig. 1



C.a.···Commissura Meynerti.
Ch. ···Chiasma nervi optici
C.M.···Commissura Meynerti
Com.m.···Commissura media
Ep.···Epiphysis cerebri

The author himself, on the contrary, considers that it may be better to suppose the so-called intermedial center for the completion of the skilful and fine ocular movement.

In view of the facts that various strange disturbances of ocular movements are found among those patients suffering from brain stem lesions, the direct connection between cortical center and the peripheral nerve nuclei can perhaps not explain those disturbances.

In the author's experiment, the place where the ocular movements were elicited cannot always be the oculo-motor center, but may be directly correlated with it, since the stimulation current seemed to be localized near the electrode tip in the author's experiment. The obtained results were summarized as follows.

Stimulus. As for the experimental results, the stimulation by 1 volt and 1 cycle per second could not elicite the ocular movements. Oculo-motor nerve root (No. 4) induced horizontal movements. Since the stimulus of 2–5 volts and 1c/s induced ocular movements without failure and with minute accompaning phenomena, it may be just the adequate stimulus. The stimulus above 7 volts made it difficult to do experimental observations.

Stimulus and laterality of the ocular movements. Binocular movements were observed in nine cases and monocular in six respectively.

In binocular cases in which the difference of the movements was noted, the homolateral movements were marked. Monocular movements were observed at the homolateral side and in three they were converted to bincolar movements with increasing stimulus intensity. In case No. 3, 6, 7, the stimulus of 8–10 volts induced monocular movements repeatedly.

Changes of the ocular movements by stimulus. The changes of coular movements were seen in six cases. At the onset of ocular movements, unstable movements occured several times and the movements are stabilized and more repeated stimuli converted the direction of the movements. It may represent the complexity of the central mechanism. Two cases of No. 7 and No. 9, may suggest the intimate spatial connection between the

two points which are correlated with horizontal and vertical eye movements.

Monocular movements. The ocular movements are equal and symmetrical on both sides (Hering's law). There are some reports on the curious, volitional seperated eye movements and also experimental reports concerning these phenomena have been done by Hitzig and Ishigooka et al..

In the author's cases, monocular movements were homolateral in all cases. Considering this, with the facts that the homolateral movements are much stronger in the binocular movement, it is very interesting. The neural mechanism of these facts is not obvious. The transition of the ocular movements with the changing stimulus intensity may represent a facilitation, the accumlative effect and the changes of the threshold might be the characteristics of the central nervous system.

Binocular movements. In binocular movements, horizontal, vertical movements, and conjugate deviation were observed. Horizontal movements were directed to the contralateral side, vertical movements were only upward without any downward movements.

Conjugate deviations were homolateral in two cases and contralateral in one. In the case of No. 9, three kinds of conjugate movements shifted mutually.

a. Horizontal conjugate movements. From the previous theory, the following places have been considered to be connected with ocular movements in the thalamus and hypothalamus from the upper end of the medial thalamic nuclei to the lower end of the Meynert's fasciculus; the place where the anterior infundibulum shifts to posterior, lateral hypothalamic nucleus around the end of the internal capsule, inferior part of the lateral hypothalamic nucleus and mid-portion between above mentioned nucleus and nucleus proprius of the cerebral peduncle, and the cerebral nucleus proprius at the latero-posterior part of lateral hypothalmic nucleus.

It is imagined that the lateral hypothalamic nucleus may be connected with horizontal ocular movements, perhaps together with the nucleus proprius of the cerebral peduncle.

b. Vertical conjugate movements. In the author's experiment, the following places had something to do with the upward ocular movements: the medial portion of the lateral hypothalamic nucleus, and the inferior part of the medial hypothalamic nucleus, the lateral part of the medial thalamic nucleus b and the inferior part of Meynert's fasciculus, the neck of the medial infundibulum, the lateral part of the medial thalamic nucleus b and the pulvinar at the peripheral part of the anterior thalamic nucleusc.

No single case of downward movements was noted.

c. Conjugate deviation. Conjugate deviation was observed in three cases. Homolateral conjugate deviation was found in the upper part of the medial thalamic nucleus and the contralateral conjugate deviation in the antero-lateral part of the mamillary body. Above mentioned points seemed to be intimately connected with the central mechanism of ocular movements. However, diencephalic structures have fine physyolgical functions and anatomical complexity.

Summary

The author observed exact and various ocular movements in cats by stimulating the diencephalon, using the isolated out-put stimulator.

1. The adequate stimulus has the intensity of 2-5 volts and a frequency of one cycle per second.

2. Binocular movements were observed in many cases and monocular movements in six cases.

3. In those cases in which the difference of movements of both eyes was noted, the homolateral movements were stronger and monocular movements were observed homolaterally.

4. Binocular movements were divided into three, horizontal, vertical and conjugate and mutual transition of these were found.

5. Horizontal movements were all directed to the contralalteral side.

6. Vertical movements were all directed upward and no downward movement was found.

7. Monocular movements were shifted to binocular with increasing intensity of the stimulus.

8. The location of the stimulating electrode tip indicates that the lateral hypothalamus and nucleus proprius of the cerebral peduncle have intimately correlated with ocular movements. Therefore the present results may not be enough for the full explanation of the central mechanism of ocular movements.

Although above experimented results will be not enough to indicate that oculomotor centers are located in the diencephalon, it may be that the described diencephalon has intimate connections with ocular movements.

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STUDIES ON THE FUNCTION OF LACRIMAL PATHWAYS

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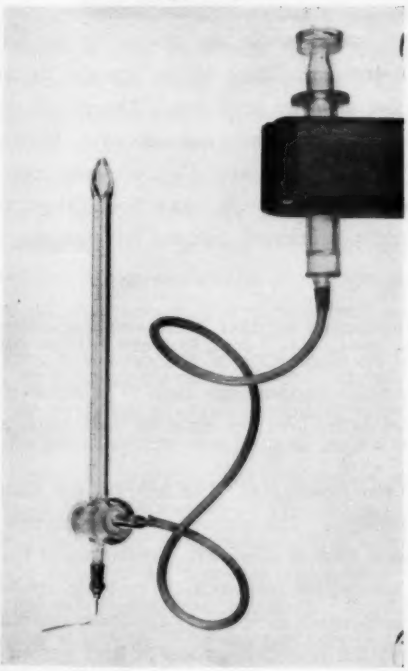
Introduction

The mechanism of the drainage of the tears has been discussed for about a century. The examination of both passage and function of the lacrimal pathway is necessary for effective treatment of epiphora. Nevertheless only the examination of passage has been done and then its method has been improved in our country. In view of the present situation, the author set to study on the function, the so-called pumping action, of the human lacrimal pathways in 1952, and got some knowledges about it. In recent years Kuribayashi observed the opening of the nasolacrimal duct by the nasolacrimaloscope devised by him, and agreed to the sac dilation theory.

Instruments for examination

Fig. 1. The watermanometer for the lacrimal pathway.



1. Watermanometer devised by the author (Fig. 1)

The apparatus consists of two parts as follows :

Pressure gauge. The glass tube marked with the scale up to 100 mm. is called " pressure tube ". It is 0.6 mm. in inside diameter, and it is larger at the upper end. This tube has a three way stop-cock nearly at the lower end. In the examination of the lacrimal sac, a 1/2 gauge injection needle of which the tip is closed and blunt, and which has a side opening within one mm. from the tip, is attached to the lower end of the pressure tube ; in the examination of the lacrimal canaliculus a 2/3 gauge needle is used and besides it is marked on the point of four mm. distant from the tip. These needles are bent at a right angle in the middle part.

Syringe. A syringe set higher than the site of the pressure gauge is connected with a branch of the latter by a rubber tube.

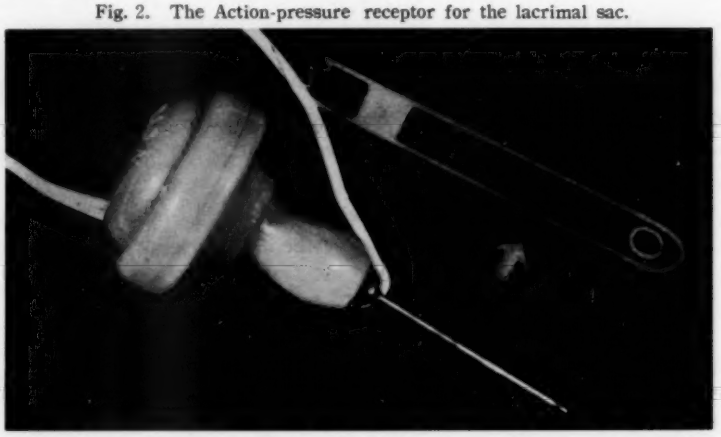
2. Unipolar needle electrode

3. Surface electrode for the lacrimal pathway

A Bowman's lacrimal probe (No. 0) which is insulated except the part extending 0.5 mm. from the tip is available.

4. Action-pressure receptor for the lacrimal sac (Fig. 2)

Fig. 2. The Action-pressure receptor for the lacrimal sac.



A crystal receiver (Rion) equipped with a 2/3 gauge needle was devised by the author and this is the equipment of the action-pressure receptor. This needle has also the same side hole that the 1/2 gauge needle, attached to the pressure tube, has. The part at which the needle is connected with the receiver is tightly sealed up with resin-paraffin. Moreover, this needle is not insulated extending about 2 mm. at the point 11 mm. away from the tip, and then this uninsulated part of the needle is to come in contact with the vertical portion of the lacrimal canaliculus, when this needle is inserted into the lacrimal sac.

Methods

Fluoresceine test. Twenty cmm. of 2% fluoresceine solution is dropped correctly and carefully in the conjunctival sac and the state of its elimination is observed and recorded every 5 minutes; it is quantity, color tone, and the disappearance time to be observed in the lower fornix. This test is always carried out to confirm the state of tear elimination before the following examinations.

Manometric examination of the lacrimal sac and canaliculus. The subjects are anesthetized locally by nupercaine and lie at supine position. The procedure of experiment by the watermanometer is as follows: The pressure tube is filled with physiological sodium chloride solution, flowing in from the syringe by turning the stop-cock. The 1/2 gauge needle attached to the pressure tube is inserted into the lacrimal sac through the lower canaliculus and fixed onto the nasal wall of the sac, and then the pressure gauge is held perpendicu-

larly. Thus, the cavity of the sac communicates with that of the syringe. In the case of chronic dacryocystitis, the cavity of the sac is washed sufficiently with pressure by the syringe. During the washing, the degree of resistance in the syringe and the existence of counter-current observed in the upper or/and lower punctum may reveal in general the grade of organic lesion in the nasolacrimal duct. Moreover, the precise state of passage in the naso-lacrimal duct is just to be found by measuring "the relative falling velocity". This method is as follows: The subject keeps the lid closed. After the cavity of the pressure gauge has been communicated with that of the sac by turning the three way stop-cock, the time that it takes for falling of the meniscus of the fluid up to 50 mm. in the pressure tube is measured and compared with that measured as control before inserting the needle into the lacrimal pathway. This ratio is multiplied by 100. This value is designated as "relative falling velocity."

And then, after drawing out the needle, the fluid in the lacrimal pathway is expelled with finger-pressure on the part of the sac and the needle is inserted into the sac again. This time, the sac is filled with the fluid flowing in spontaneously from the syringe without pressure. The speed in falling of the meniscus of the fluid in the pressure tube increases in proportion to the diameter of the nasolacrimal duct, when the communication between the sac and the pressure tube is opened by turning the three way stop-cock. In the cases of the completely obstructed nasolacrimal duct, the meniscus can not fall. And then the subject is told to do normal palpebral movements, that is, closing and opening of the lids and blinking. Just then the vertical shaking movement of the meniscus is observed and the range of this movement is recorded; 1 mm. in the scale of the pressure tube is equivalent to 0.3 cmm. of the fluid. Thus the pumping quantity is revealed, that is, the falling of the meniscus shows the suction quantity and its rising shows the expulsion quantity.

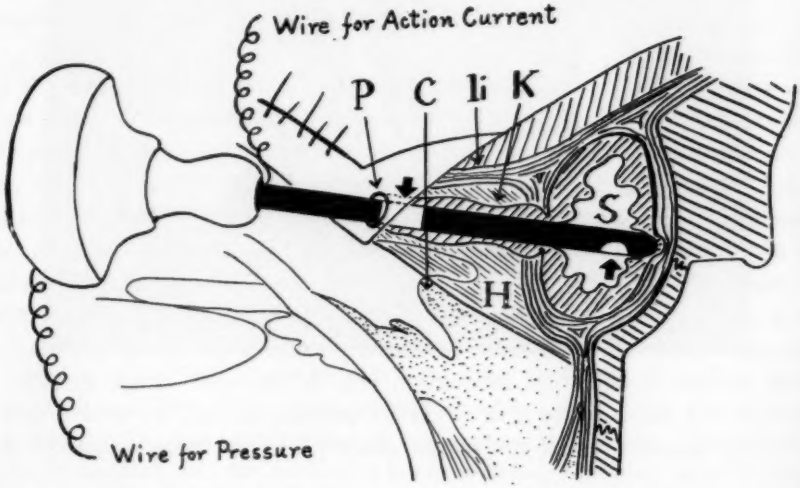
In the examination of the canaliculus, the 2/3 gauge needle attached to the pressure gauge is inserted into the lower canaliculus up to 4 mm. from its tip so far as to reach the beginning part of the horizontal portion of the canaliculus, and then the needle introduced to the vertical portion of the canaliculus is swung round so as to be level with the horizontal portion. The subsequent examinations, that is, the observation of the pumping action and the measurement of its power are conducted by the same method as in the sac. But the pumping power measured like this may only reveal that in the horizontal portion. So the pumping power or pumping quantity in the whole lower canaliculus is calculated correctly by the fact that its total length is 10 mm. (Ashikaga).

Electrical recordings in the action of the lacrimal portion of the orbicularis oculi muscles (Horner's muscle). The subject who had normal lacrimal pathways was locally anesthetized with nupercaine. The unipolar needle electrode was stuck into Horner's muscle through the lacrimal caruncle or inserted into the vertical portion of the lower canaliculus through the punctum. Furthermore the surface electrode was applied by inserting it into the lacrimal pathway in the same procedure such as probing. And thus

action potentials in Horner's muscle produced while blinking were recorded with the Braun tube oscillograph.

Electrical simultaneous recordings of the action of Horner's muscle and of the pumping action in the lacrimal sac. The subject who had normal lacrimal pathways was locally anesthetized by nupercaine. The action-pressure receptor was inserted into the lacrimal sac through the lower canaliculus as shown in Fig. 3: The needle tip of the receptor

Fig. 3. The Action-pressure receptor inserted into the lacrimal sac (c. f. Fig. 2). S: Lacrimal sac, P: Lower punctum, C: Caruncle, li: Medial palpebral ligament, K: Lower canaliculus, H: Horner's muscle.



was fixed onto the nasal wall of the sac so that the side hole of the needle might face towards the posterior wall. Two wires were fixed on the part of the cheekbone by sticking plaster. And thus, while blinking, both action potential in Horner's muscle and potential of the receiver which was produced by the pressure changes in the lacrimal sac were led and recorded simultaneously by the electroencephalograph. Action potential was led from the vertical portion of the canaliculus in which it was to be led most easily as mentioned later.

On the other hand, some difficulties were pointed out in identifying the action of the lacrimal canaliculus in the pumping action recording experiment by the other special receptor.

Materials and Results

In the examination of the sac the meniscus in the pressure tube rises usually at the openingmovement of the lids and falls at closing; on the other hand in the canaliculus, it falls at opening and rises at closing.

Fig. 4. The EMG of Horner's muscle under the unipolar lead in a normal subject. P: Lower punctum, C: Caruncle, li: Medial palpebral ligament, H: Horner's muscle, S: Lacrimal sac.

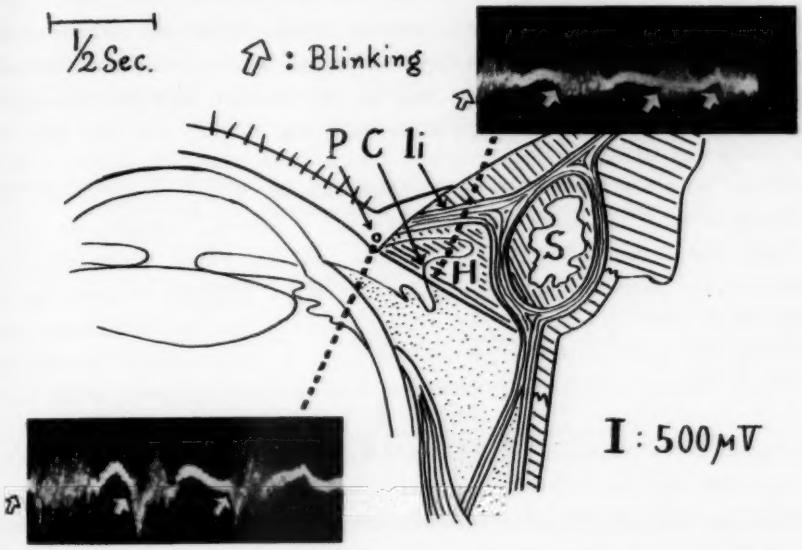
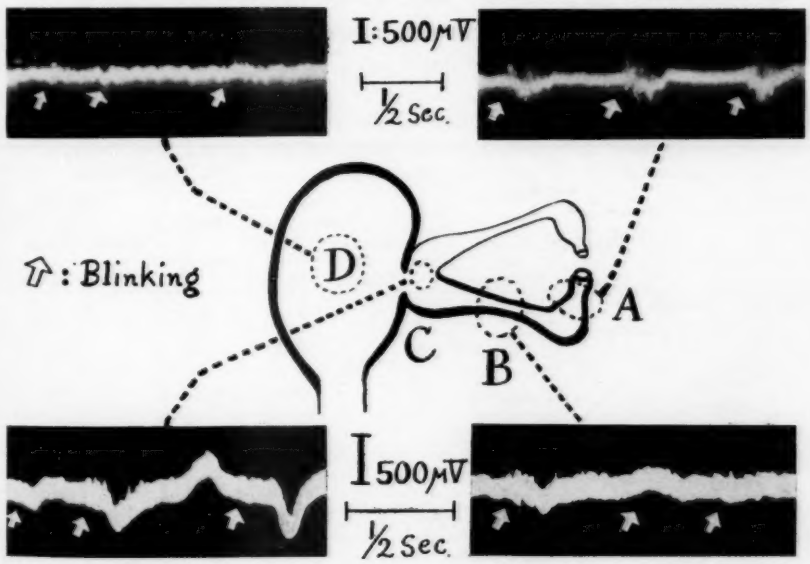


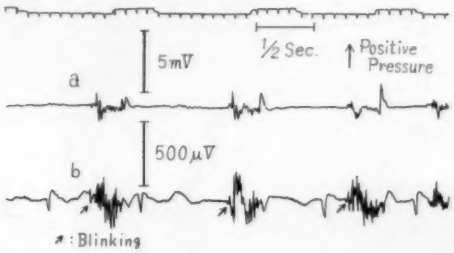
Fig. 5. The EMG of Horner's muscle under the surface lead in a normal lacrimal pathway. A: Vertical portion of lower canaliculus, B: Horizontal portion, C: Sinus of Maier, D: Posterior wall of sac.



The EMG of Horner's muscle is shown in Fig. 4 and Fig. 5: Each spike discharge appears in the phase of closure while blinking and it can be recorded most clearly in the vertical portion of the canaliculus.

In the simultaneous recording of the pressure changes in the sac and the spike discharge of Horner's muscle, the negative pressure changes were recorded simultaneously with the appearance of the spike discharge and on the contrary, with its disappearance the positive pressure changes were recorded as shown in Fig. 6.

Fig 6. The pressure changes in the lacrimal sac (a) and the EMG of Horner's muscle (b) recorded simultaneously in a normal subject (c. f. Fig. 2 & 3).



Fluoresceine solution dropped in the conjunctival sac is carried away completely within 25 minutes in normal cases. So the cases in which fluoresceine solution remained in the lower fornix over 25 minutes were selected as pathological subjects suffering from epiphora, and they were commonly getting chronic dacryocystitis and complete or incomplete obstruction of the nasolacrimal duct.

The watermanometer test was carried out in about a hundred lacrimal sacs and canaliculi as shown in Table 1.

Table 1. The Probative Rate and the Power of the Lacrimal Pump

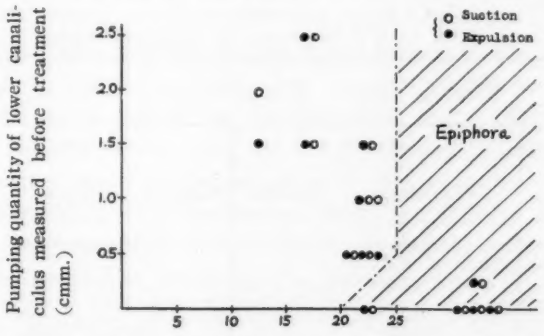
		NORMAL			PATHOLOGICAL					
					Obstruction of Nasolacrimal Duct			Chronic Dacryocystitis		
		Number of Cases	Range of Values	Mean	Number of Cases	Range of Values	Mean	Number of Cases	Range of Values	Mean
Lacrimal Sac	Relative Falling Velocity	40	50~200	115	35	0~40	6	27	0~40	3
	Expulsion (cmm.)		0~3.3	0.3		0~0.6	0.1		0~0.9	0.1
	Suction (cmm.)		0~4.5	0.5		0~0.6	0.1		0~0.9	0.1
	Presence of Pumping Action		55%			31.4%			18.5%	
Lower Canaliculus	Expulsion (cmm.)	31	0~5.0	1.5	35	0~5.0	1.2	27	0~3.5	0.4
	Suction (cmm.)		0.5~5.0	1.7		0~5.0	1.3		0~3.5	0.5
	Presence of Pumping Action		100%			94.3%			48.1%	

Generally it was found in normal cases, that the greater the " relative falling velocity " was, the less the pumping action of the sac. Six cases out of normal sacs showed the reverse pumping action and the meniscus rose at the closing movement of the lids and fell at opening. In the tests of normal canaliculi 4 cases also revealed the reverse pumping action ; the meniscus fell at the closing movement of the lids and rose at opening.

In each pathological subject, the presence of the involvement in the passage of the nasolacrimal duct was confirmed by the remarkable decrease of the relative falling velocity. Generally speaking, the pumping quantity of the sac was found to be decreased or abolished in both chronic dacryocystitis and obstruction of the nasolacrimal duct. On the other hand, generally the pumping quantity of the canaliculus was found not to be decreased in cases of obstruction of the nasolacrimal duct, but in chronic dacryocystitis it was usually proved to be decreased or abolished. Five lacrimal sacs and no canaliculus in these pathological subjects showed the reverse pumping action. The pumping quantity in the pathological subject was not always related to the period during which he had been suffering from epiphora, and no pumping action was observed, in spite of the short duration of the involvement, in some lacrimal sacs.

Moreover, the efficacy of the treatment with vinyl-tube method was investigated by the above mentioned test in 34 cases, including 11 cases of chronic dacryocystitis and 23 cases of obstruction of the nasolacrimal duct. These results were as follows: It was not so easy to improve an obstructed nasolacrimal duct with this treatment, that is, the relative falling velocity was not always able to become more than 50 and only in 13 cases it improved. In these cases, the relation between the state of tear elimination revealed with the fluoresceine test and the pumping quantity of the lower canaliculus is shown in Fig. 7. Finally, complete subjective and objective disappearance of epiphora was seen in 8 cases out of these 34 cases, that is, in only 24% of the cases. And yet it should be emphasized, that

Fig. 7. The state of tear elimination in subjects in which obstruction of their nasolacrimal duct has been improved by the vinyl-tube method.



Disappearance time of fluoresceine recorded after treatment (min.,)

the pumping action of the lacrimal sac was found to be abolished completely in 5 cases out of those improved 8 cases.

Discussion

The watermanometer test devised by the author may be a kind of load test, but hydraulic pressure which is equivalent to the elevation of the head of the fluid column in the pressure tube may be considered to have only slight effect on the wall of the lacrimal pathway. So the vertical movements of the meniscus in the pressure tube caused by the palpebral movements may reveal the so-called lacrimal pump (Jones), and the pumping quantity thus measured represents numerically the function of the lacrimal pathway.

Moreover, the author has succeeded in recording the electromyogram of Horner's muscle which is conclusively considered to give rise to the lacrimal pump, and has been able to observe simultaneously both action potential of this muscle and the pumping action of the lacrimal sac. The negative pressure in the sac was demonstrated simultaneously with the contraction of Horner's muscle while blinking. This fact seems to support the sac dilation theory (Schirmer). Besides, by the fact that the spike discharge of Horner's muscle is able to be found only at closing movements of the lids and not at opening, this muscle is proved to be a part of the palpebral fissure constrictor as emphasized by Schirmer.

Some workers (Ashikaga, Fazakas, and others) who support the sac dilation theory have described, that the canaliculi also are extended by the contraction of the orbicularis oculi muscle. On the other hand, other workers (Benjamins & Rochat, Frieberg, and others) who uphold the sac compression theory have reported, that the canaliculi are also compressed by swelling of the oribicularis oculi fibres being constricted at closing movements of the lids. The results in the experiment performed by Rosengren & Kugelberg also give support to the latter.

The mechanism of tear elimination is to be considered as shown in Table 2, according

Table 2. The Mechanism of Tear Elimination

Blinking	Closing Phase	Internal Pressure	Opening Phase	
Horner's Muscle	Contracts		Relaxes	
Lacrimal Canaliculi	**Compressed**	**Positive**	**Negative**	**Dilated** by its own Elasticity.
Lacrimal Sac	**Dilated** (Lateral Wall is pulled upon.)	Negative	Positive	**Compressed** by its own Elasticity.
Counter-current is hindered in	Puncta		Sinus of Maier	

to the author's experimental results: The tears may be sucked in the canaliculi at the opening phase while blinking, and its further conduction into the sac may be accomplished not so much by sucking action of the sac as by expulsive action of the canaliculi at the closing phase of the lids.

The function by which the counter-current seems to be hindered by the valves in the sinus of Maier appears not to be needful so much, because the pumping action of the sac usually is not so powerful, compared with that of the canaliculus. Consequently, the reverse pumping action of the canaliculus observed in 4 cases of normal subjects is to account for as follows: If the pumping action of the sac is more active and powerful than that of the canaliculus, it is not strange that such a phenomenon should appear, because the inner pressures in the sac overcome that in the canaliculus. On the other hand, the reverse pumping action of the sac observed in 6 cases of normal subjects seems to appear on account of the feebleness of the medial palpebral ligament, because if this ligament is feeble, the raised intraorbital pressure at the closing movement of the lids comes to compress the sac.

From the fact mentioned above, it seems justified to conclude, that tear elimination is carried out by the alternating actions of the lacrimal sac and the canaliculi, namely by such an active movement as peristalsis. Krehbiel also seems to support such an opinion. Ōtsuki has proved the contrary from the anatomical point of view that Horner's muscle is not found to arise from the part of the sac, and he has stated, that it is a mistake that Horner's muscle may dilate the lacrimal sac to suck the tears into it as described in some textbooks. In recent years, however, Jones has modified the anatomy of the palpebral muscles based on his excellent work, and asserted, that the lacrimal sac should be provided with negative pressure by "the deep heads of the preseptal muscles" which arise from the lacrimal diaphragm.

Regarding the diagnostic aid in obstruction of the nasolacrimal duct, the author's method measuring the relative falling velocity is believed to be better than the others, that is, fluoresceine dye test, irrigation, probing, or radiography. The variations of the relative falling velocity in normal subjects are considered to be related to the individual variations of physiological inside diameters of the nasolacrimal duct (Ashikaga, Aoyama et al.). The value of the relative falling velocity in pathological subjects is very small, that is, the passage of these nasolacrimal ducts is very poor, so the pumping action of the sac in these subjects should be proved in the pressure tube more readily than in normal subjects, but this pumping action, none the less, is inferior to that in normal subjects. Therefore, the pumping action of the sac shows to be considerably decreased in chronic dacryocystitis and obstruction of the nasolacrimal duct. This is likely to be suggested by the following fact: No canaliculus in pathological subjects shows the reverse pumping action which is to be observed in some cases of normal subjects.

The probative rate of the pumping action of the sac is lower in cases of chronic dacryocystitis than in cases of obstruction of the nasolacrimal duct. This fact seems to

suggest, that the function of the sac under chronic dacryocystitis is apt to be remarkable disturbed by the inflammatory lesion. Besides, the function of Horner's muscle which causes the pumping action may be involved by its slackening accompanied with the atony of the sac. Such a disturbance of movement in Horner's muscle is considered also to be related to the fact, that the pumping action of the canaliculus in cases of chronic dacryocystitis is much more decreased as compared with that in cases of obstruction of the nasolacrimal duct. The reverse pumping action of the sac observed in 5 cases of pathological subjects is believed to occur due to the atony of the sac in which the sac may directly receive the raised intraorbital pressure at the closing movement of the lids.

The results of the treatment with vinyl-tube method give rise to the conclusions that the good function of the lacrimal sac is not always necessary for the conduction of the tears and the function of the lacrimal canaliculus plays a much more important role in it. And further, the tears which have been conducted into the sac from the canaliculi can be also eliminated in the nasal cavity through the nasolacrimal duct by gravitation only, as asserted also by Frieberg; when this duct is sufficiently wide, that is, the relative falling velocity is more than 50. The lower canaliculus is generally recognized to be more important than the upper one in tear elimination. Consequently, the results in the author's experiment show that the treatment for the obstructive nasolacrimal duct may be expected and effective only when the pumping quantity of the lower canaliculus is more than 0.5 cmm., and that the lower lacrimal punctum must not be slit in the treatment of epiphora, because the pumping action of the lower canaliculus will be damaged.

The cause of obstruction of the nasolacrimal duct or chronic dacryocystitis remains still to be explained well, but at least the basic factor for its onset is believed to be as follows:

The marked decrease of the function of the lacrimal sac may not be the result of organic lesion in the nasolacrimal duct but its cause, because the pumping quantity, as described above, is not always related to the period during which the patient has suffered from epiphora and in some subjects, the function of their lacrimal sacs has been already damaged in spite of the short duration of the involvement. Such a weakness in the function of the sac may result in some congenital latent defect in its wall or Horner's muscle. In the lacrimal pathway, having the weak pumping action of the sac, the tears are only conducted to the nasal cavity through the nasolacrimal duct mainly by gravitation. When such an unfavorable conduction of the tears, even though there is no complaint of epiphora, continues as it is for a long period, the pathogenic organisms coming from the conjunctival sac or the nasal cavity are apt to stagnate and anchor in the sac and nasolacrimal duct. As the result, the sac and duct give rise to inflammatory lesion, and the function of the sac becomes damaged more and more. Thus finally a vicious circle comes to take place, and the nasolacrimal duct comes to be obstructed by the organic lesion, as if the ditch in which sewage flows slowly is frequently choked with debris. And then chronic dacryocystitis may develop consecutively.

Therefore, chronic dacryocystitis or obstruction of the nasolacrimal duct will frequently recur sooner or later, even if the nasolacrimal duct becomes wide and passable with the treatment. In the treatment, therefore, the lacrimal sac should be widely opened directly to the nasal cavity, disregarding the nasolacrimal duct, when the function of the lower lacrimal canaliculus is proved to be kept up well. This is just the reason why dacryocystorhinostomy has come to be recommended in recent years. In cases of long standing chronic dacryocystitis, however, dacryocystectomy might still be an only treatment, because the function of the lower lacrimal canaliculus as well as the sac might be considerably or completely damaged.

Conclusion

1. Tear elimination is accomplished by a kind of peristalsis, that is, alternating movements of the lacrimal sac and the canaliculi. Moreover, the canaliculi have a much more important role in the conduction of the tears.

2. The author agrees to Schirmer's opinion that Horner's muscle which causes the pumping action of the lacrimal pathway constitutes a part of the palpebral fissure constrictor. And the pumping action of the lacrimal sac occurs simultaneously with the movement of Horner's muscle.

3. The pumping action of the lacrimal sac is proved to be decreased or completely disappeared in both chronic dacryocystitis and obstruction of the nasolacrimal duct.

4. On the other hand, it is proved, that the pumping action of the lower lacrimal canaliculus is little involved in the cases of obstruction of the nasolacrimal duct, but in the cases of the chronic dacryocystitis it is considerably or completely damaged.

5. The onset of obstruction of the nasolacrimal duct or chronic dacryocystitis may be based on some disposition for the involvement in the pumping action of the lacrimal sac, and this disposition seems to be related to the original weakness of the pumping action founded as individual variations.

6. As to the treatment for organic lesion of the nasolacrimal duct or lacrimal sac, dacryocystorhinostomy should be indicated, if the function of the lower lacrimal canaliculus is proved to remain still, that is, the pumping quantity is more than 0.5 cmm.

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BIOCHEMICAL STUDIES ON THE PLASMOID AQUEOUS
REPORT III NONPROTEIN NITROGEN AND FREE AMINO ACIDS

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Introduction

The formation of the aqueous humor has been the subject of extensive studies for many years, and main theories proposed are the ultrafiltration[1]-and secretion-diffusion[2] theory. However, the real mechanism of the aqueous formation is still in dispute. In order to know this mechanism, it is of particular importance to make analytical studies on the chemical constituents of the plasmoid aqueous. However, detailed study on the time course of the change in chemical composition of the plasmoid aqueous, after the withdrawal of the aqueous humor, is still rarely met with, although such a study would have an outstanding value to solve the problem. The studies on the time course should, of course, be made for many constituents of the aqueous humor.

In the series of the previous investigations, the author studied on the time course of the change of some constituents of the plasmoid aqueous. The total protein increased immediately after the withdrawal of the aqueous humor[3]. In this case, the increase of albumin preceded the increase of globulin. Another peculiar feature of the change was recognized for potassium[4]: A remarkable increase of potassium was found to occur immediately after the withdrawal of the aqueous humor. On the contrary, the amount of sodium exhibited no remarkable change.

In recent years, a new method of high voltage paper electrophoresis, using the horizontal position of filter paper, was established in the Biochemical Department of Tokyo Medical and Dental University[5]. This method enables to isolate free amino acids accurately and distinctively. In consequence, the evaluation of the quantitative change of amino acids can be made with ease and accuracy.

In the present investigation, therefore, the time courses of the change of amount were studied for nonprotein nitrogen (N.P.N.) and free amino acids of the plasmoid aqueous of rabbit at several intervals after the withdrawal of the aqueous humor. Then it was found, that these constituents underwent a selective permeation in the period shortly after the escape of the aqueous humor. On the basis of these experimental results, some discussions were made on the mechanism of aqueous formation.

Materials and Methods. Adult albino rabbits, weighing about 2 kg. were used, after

they were fed on nothing at least for 24 hours.

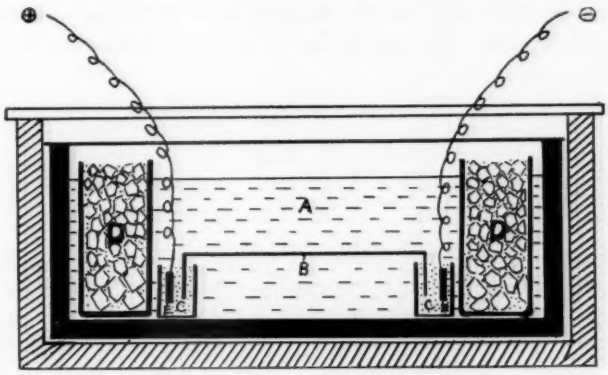
The plasmoid aqueous was withdrawn using a syringe, under no anesthesia, at intervals of 10, 15, 30 minutes, 1, 1.5, 2, 4, 6, 7, 10 and 24 hours after the withdrawal of the normal aqueous humor. The aqueous humor obtained from 4 eyes was pooled and used for the measurements described below.

The blood was also withdrawn from the auricular vein of the same rabbits; the serum was isolated therefrom and was subjected to the following measurements.

Determination of the concentration of N.P.N. The aqueous humor obtained was devided into 2 parts; the first part was used to determine the concentration of total nitrogen and the other for N.P.N. after deproteinization with trichloracetic acid. To determine the concentration of nitrogen, the micro-Kjeldahl method was employed.

Isolation of free amino acids and the density distribution ratio. The horizontal type of the apparatus for the high voltage paper electrophoresis (using the horizontal position of filter paper) was employed to isolate free amino acids. This apparatus was, in principle, composed of a rectifier, which could supply 12 kilovolts, and a box for electrophoresis

Fig. 1. The box for electrophoresis.



(Fig. 1). The box was filled with hexan (A), which was cooled with ice or solid carbon dioxide (D) during the procedure. This box was also equipped with two buffer troughs (C), in which a buffer composed of formic acid 5 cc., acetic acid 15 cc. and aq. dest. 80 cc. was filled. A filter paper strip (B) moistened with the buffer was placed on a supporting frame in this box, both ends of the filter paper strip being soaked into the buffer. On the original line drawn on the anodal side of the filter paper, the sample of the aqueous humor was smeared as homogenous as possible. And then the potential difference of 4 kilovolts, which corresponded to the potential gradient of 111v/cm., was applied, by means of platinum electrodes (E), to both ends of the filter paper strip for 20 minutes.

When the electrophoresis ended, the filter paper strip was dried rapidly and stained with 0.2 per cent ninhydrin solution. Then this filter paper was made to be translucent

by immersing it into warmed paraffin.

The density of colored bands of the filter paper, each representing an amino acid fraction, was measured by means of an electronic densitometer for filter paper (devised by Kobayashi), using a green filter (550 mμ), and a pattern of density curve was obtained. The area of the density curve, corresponding to a given fraction, was measured with a planimeter, and the ratio of this area to the total area of the pattern was calculated. This ratio, expressed in per cent, was called the density distribution ratio of the given fraction. The density distribution ratios were computed for 8 fractions isolated. By the use of the density distribution ratio, the evaluation of the change of the relative amount of each fraction could be made, although it was impossible to determine its absolute concentration.

Results

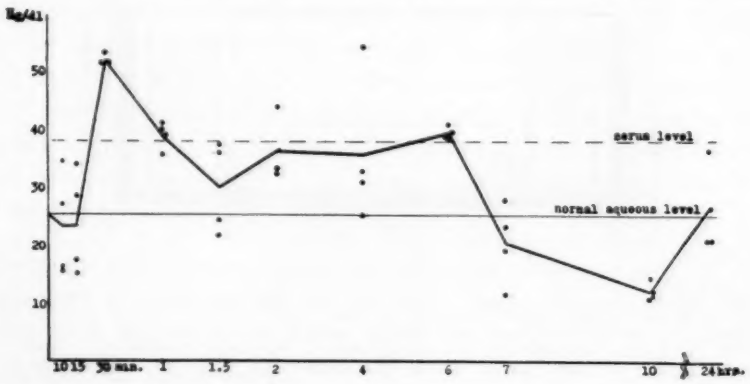
1. Total nitrogen and N.P.N. in the normal aqueous humor and blood serum

The average concentration of total nitrogen was 38.8 mg/dl in the aqueous humor and 1017 mg/dl in the serum. On the other hand, the concentration of N.P.N. averaged 25.5 mg/dl in the aqueous humor and 38.5 mg/dl in the serum.

2. N.P.N. in the plasmoid aqueous

The concentration of N.P.N. in the plasmoid aqueous averaged 23 mg/dl at intervals of 10 and 15 minutes after the withdrawal of the aqueous humor (Fig. 2). This value

Fig. 2. N.P.N. of plasmoid aqueous. line: time course of the average concentration.



was far lower than the normal aqueous level. However it increased thereafter and the maximum concentration, the average being 52 mg/dl, was reached at an interval of 30 minutes. In the following period, a gradual decrease of N.P.N. was observed; and then, an abrupt decrease to a level far below that of the normal aqueous level occurred at an interval of 7 hours, and it did last till the interval of 24 hours when the normal aqueous level was restored.

3. Free amino acids in the normal aqueous humor and blood serum

Eight fractions of free amino acids could always be isolated from the aqueous humor. According to the facility of mobilisation, these fractions were designated successively as I. II, III, IV…VIII fractions. When the distance of the band of a given fraction from the original line of electrophoresis was divided by the distance of I fraction, the ratio of mobilisation of this fraction was obtained. This ratio of mobilisation is a characteristic constant factor of each amino acid. In the present experiment, the ratios of mobilisation of 8 fractions were strictly in accordance between serum and aqueous humor. Therefore, it is reasonable to conclude that the pattern of free amino acids in the aqueous humor is the same as that in the serum. By the preliminary experiment[6], these fractions were identified as shown in table 1. The ratio of mobilisation and the density distribution ratio

Table 1. Amino Acids in Aqueous Humor and Serum

Fraction	Amino acid	Ratio of Mobilisation	Density Distribution Ratio (%)	
			Aqueous	Serum
I	Lysine	1.00	12.7	14.8
II	Histisine / Arginine	0.93	7.3	4.5
III	Glycine	0.82	9.8	21.5
IV	Alanine	0.73	14.7	14.9
V	Valine	0.61	15.3	12.4
VI	Leucine	0.57	6.3	6.7
VII	Phenylalanine	0.52	22.9	17.6
VIII	Glutamic acid	0.42	10.3	7.2

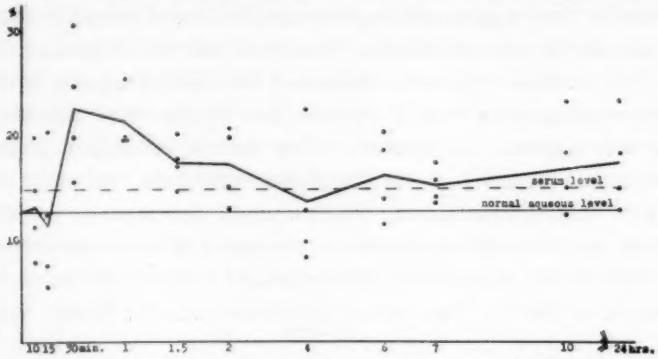
of each amino acid fraction both in aqueous humor and in serum are also given in this table. As readily seen in the table, considerable difference of the density distribution ratios of amino acids was found between serum and aqueous humor.

4. Free amino acids in the plasmoid aqueous

The ratio of mobilisation of each amino acid fraction in the plasmoid aqueous was equal to that of the amino acid in the normal aqueous humor and serum. Therefore, the pattern of the amino acids in the plasmoid aqueous is the same as that in the normal aqueous and serum. The density distribution ratio of each amino acid fraction was obtained at several intervals after the withdrawal of the aqueous humor. The results will be described for individual fractions.

I fraction(Lysine). At intervals of 10 and 15 minutes, the density distribution ratio of lysine of the plasmoid aqueous was almost at the normal aqueous level, which was lower than the serum level (Fig. 3). However, at the interval of 30 minutes, a marked increase beyond the serum level took place, and then it decreased gradually to the serum level, which was maintained until the interval of 24 hours.

Fig. 3. Lysine of plasmoid aqueous. line: time course of the average concentration.



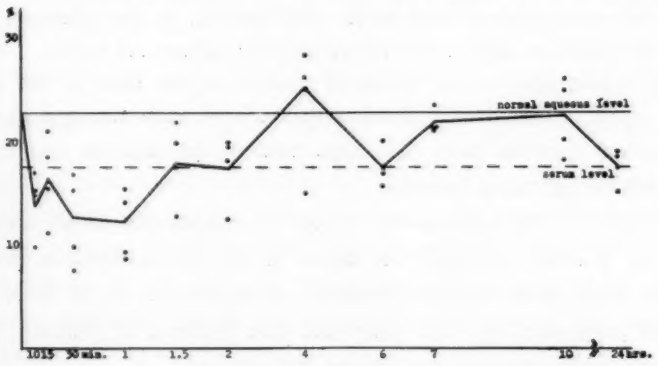
II fraction (*Histidine and/or Arginine*). The density distribution ratio of this fraction in the normal aqueous was higher than that in the serum. At inteavals of 10 and 15 minutes, the density distribution ratio of this fraction in the plasmoid aqueous exhibited no remarkable difference from that of the normal aqueous. However, it decreased to the serum level, at intervals of 30 minutes and 1 hour; then it showed gradual increase and the normal aqueous level was restored after 24 hours.

III fraction (*Glycine*). In the initial period following the withdrawal of the aqueous humor, the density distribution ratio of this fraction was almost at the serum level, which was higher than the normal aqueous level. Then it decreased gradually to the normal aqueous level after 24 hours.

V fraction (*Valine*). This fraction showed a considerable decrease far down to the serum level at intervals of 10, 15, 30 minutes and 1 hour. Then it restored, after 2 hours, to the normal aqueous level, which was maintained till the interval of 24 hours.

VII fraction (*Glutamic acid*). Immediately after the withdrawal of the aqueous humor,

Fig. 4. Glutamic acid in plasmoid aqueous. line: time course of the average concentration.



this fraction decreased remarkably to a level far lower than the serum level, which was also much lower than the normal aqueous level (Fig. 4). Then it increased to the normal aqueous level after 10 hours, showing some fluctuations.

IV (Alanine), VI (Leucine) and VIII (Phenylalanine) fractions. Between the aqueous humor and the serum, there were no remarkable differences of the density distribution ratios of these fractions; these fractions underwent no remarkable change of the density distribution ratios, due to the withdrawal of the aqueous humor.

Discussion

One of the previous investigations confirmed that the withdrawal of the aqueous humor resulted in an increase of protein content of the plasmoid aqueous, which lasted for about 2 hours, the maximum concentration of protein being obtained at an interval of 1 hour. In this case, the increase of albumin preceded that of globulin with an interval. Such a lag of the transfer of globulin was also found by Tomikawa[7] for the transfer of serum protein through the glomerular membrane of the kidney in toads. Therefore, the preferential transfer of albumin can be regarded as a common phenomenon for the transfer through the biological membrane and also can be attributed to the fact that the size of albumin molecule is smaller than that of globulin. Judging from these facts, it can be postulated that the common mechanism of the transfer through the biological membrane also underlies the process of the aqueous formation.

As for the electrolytes of the aqueous humor, the potassium exhibited a peculiar behavior. In the initial period, after the withdrawal of the aqueous humor, a remarkable increase of potassium took place, and then a decrease followed. This phenomenon could not be explained by the random permeation of serum constituents into the chamber.

In the present investigation, the concentration of N.P.N. decreased far below the normal aqueous level in the initial period following the withdrawal of the aqueous humor, although an increase followed thereafter. The concentration of N.P.N. in the serum is much higher than in the normal aqueous humor. Therefore, if the plasmoid aqueous is formed by a random permeation of the serum constituents, the N.P.N. of the plasmoid aqueous should increase due to the withdrawal of the aqueous humor. Contrary to this expectation the present experiment proved an occurrence of a decrease of N.P.N. of the plasmoid aqueous, instead of increase. Moore[8] and co-workers studied on the time course of the change in the amount of the urea of plasmoid aqueous, and they obtained the results similar to those of the present author. This result can also hardly be explained by the random permeation of serum constituents, as in the case of potassium.

The qualitative identification of free amino acids and the quantitative determination of their concentration in the aqueous humor and serum were made, using paper chromatography, by Watton[9] et al. and Kinugasa[10] et al.; but their results are far from being uniform, because no accurate method of quantitative analysis has been established hitherto. In this experiment, therefore, the attempt to determine the absolute concentration

was abandoned. However, the use of the density distribution ratio was sufficient to investigate the change of the relative amount of free amino acids.

As for the time course of the change of the amino acid content of the plasmoid aqueous, individual fractions did not always reveal the same pattern of the change, when the period shortly after the withdrawal of the aqueous humor was taken into account. In case of lysine, the normal aqueous level was maintained in the initial period and an increase toward the serum level followed after a lag period. On the contrary, glutamic acid revealed a immediate decrease to a level far lower than the serum level.

In the period following the initial change, mentioned above, the concentration of amino acid fractions exhibited such a tendency that it approached gradually the serum level up or down, regardless of the direction of initial change.

When the chemical composition of the plasmoid aqueous is controled only by the random permeation of the constituents of serum, the concentration of free amino acids should inevitably approach the serum level immediately after the withdrawal of the aqueous humor. However, the present results were incompatible with this assumption. This fact indicates that the amino acids are permeated selectively into the plasmoid aqueous.

In the previous report on the potassium of the plasmoid aqueous, the author stressed that the initial increase of the potassium might be explained by a release of potassium contained within cells of the ciliary body; because the potassium is, in general, richly contained within cells, while its concentration in the serum is very low. Although there is no secretory cell, in the strict sense, in the ciliary epithelium, this release of potassium suggests, that the activation of the function of the ciliary epithelium, like secretory function, was elicited by the withdrawal of the aqueous humor. The present studies also confirmed the presence of the selective permeation of N.P.N. as well as of amino acids in the period shortly after the withdrawal of the aqueous humor. The period of the occurrence of this selective permeation was in good accordance with the period of increase of potassium. The results of the present studies, therefore, may be regarded to support the assumption just mentioned.

Summary

The time course of the change of the concentration of N.P.N. and free amino acids in the plasmoid aqueous, after the withdrawal of the aqueous humor, has been studied with following results.

1. Nonprotein nitrogen of the plasmoid aqueous decreased, compared with normal aqueous level, in the period shortly after the withdrawal of the aqueous humor. Then increase to the serum level followed.

2. The time courses of the change of the amount of free amino acids did not reveal the same pattern of the change, in the early period. In the period following the initial change, however, the density distribution ratios of amino acids showed a tendency to aproach the serum level.

3. From these experimental results, it was concluded that nonprotein nitrogen and

amino acids of the serum permeated selectively into the chamber, in the period shortly after the withdrawal of the aqueous humor.

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ZONE ELECTROPHORETIC STUDIES ON THE SOLUBLE PROTEINS OF THE CRYSTALLINE LENS

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Separation of soluble proteins from a small amount of lens material has been strenuously continued by many investigators without complete success. It is the purpose of this paper to report on the application of zone electrophoresis on a starch column to the separation of the soluble lens proteins of normal and cataractous eyes. In addition, changes in the content of each protein fraction were studied throughout the stage of development of the experimental cataract.

Method

Apparatus and Material The apparatus used for the electrophoretic separation of proteins was the zone electrophoresis apparatus of the vertical type devised by Dr. Naono. Quantitative analysis of protein was done with the tube fractionated after electrophoresis with Folin-Ciocarteau's method. The protein concentration of each fraction is plotted against the test tube number (This curve is called to be an electrophoretic pattern.).

The materials used for separation and purification of proteins were a normal bovine lens obtained as quickly as possible after death of the animal. Normal rabbit eyes were also used. The materials used in the case of experimental cataract were obtained from naphthalene cataractous eyes. In this experiment, protein solution was prepared following Fromageot's methods.

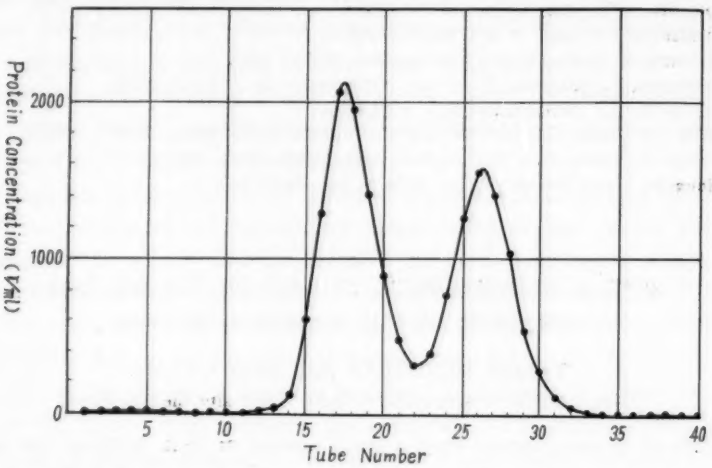
Results

I. Separation and Purification of Soluble Proteins of a Bovine Lens

Protein of the bovine lens cortex. The lens material used for the separation was pre-

pared from the superficial layer of the cortex after removal of the lens capsule. A zone electrophoretic pattern of protein aqueous solution of the lens cortex is illustrated in Fig. 1. It was revealed that the protein aqueous solution of the lens cortex is made up of the following two fractions of different mobility: the first component with the mobility of 14.3

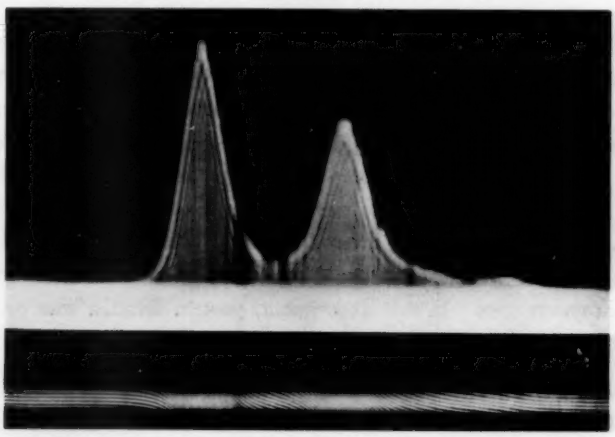
Fig. 1. Zone electrophoretic pattern of soluble protein of bovine lens cortex.



Hight of initial zone 23 cm
applied sample 26.1 mg

1/10 M Veronal-HCl buffer pH 8.6 μ=0.1
450V. 20mA 19 hours
Each fraction 1.8 ml.

Fig. 2. Electrophoretic pattern of soluble protein of bovine lens cortex.
(Spinco Analysis Model H) (Ascendens)



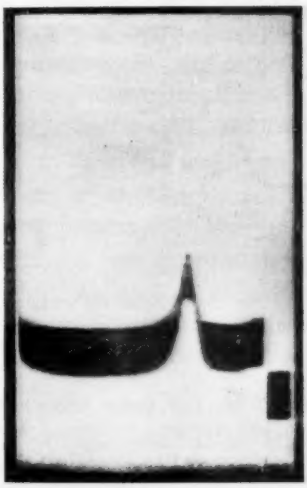
Buffer: M/10 veronal-HCl pH=8.6 μ=0.1, Dialysis 24 hours (2°C), Blade Angle 90°, 3mA, 133.4 min.

cm and the second fraction with the mobility of 9.7 cm from the initial zone in the starch column. Electrophoresis was performed in the 1/10 M veronal-HCl buffer system at pH 8.6 and μ=0.1. This electrophoretic pattern corresponds to that of the same material with the Spinco Analysis Model H. (Fig. 2)

Therefore, this zone electrophoretic method is considered to be a new additional technique to the separation of the lens protein because of its high sensitivity with which soluble cortex proteins are separated into the two fractions successfully.

Next, a question arises as to whether these fractions are made up of a purified single component or the mixture of some components. In order to clarify this problem, ultracentrifugative analysis by means of the Spinco Analysis Model E was done with the fraction tube which fell on the two peaks of the electrophoretic pattern.

Fig. 3. Pattern of ultracentrifugative analysis of a fraction with high mobility in the zone electrophoresis. (Spinco Analysis Model E)

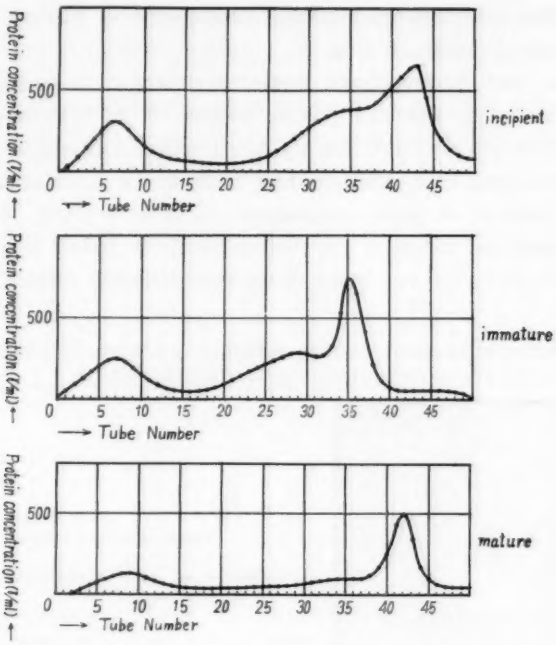


59800r.p.m. M/10 veronal-HCl buffer.
pH 8.6 32 min.

As is shown in Fig. 3, it was disclosed that these protein fractions had been so perfectly separated and purified as to be considered to be made up of a single component.

Precipitation reactions at the isoelectric point of the two fractions were also studied. A series of standard pH solutions from pH 3.0 to pH 6.6 at 0.2 pH difference were prepared with acetic acid. Three ml. of these standard solutions was placed into the test tubes, into which 0.1 ml. of protein solutions were added. Final pH was determined with the test tubes which produced turbidity. In addition, turbidities were measured of these tubes at the wave length of 450 mμ. In this method, the fraction with high mobility showed the highest turbidity at pH 5.2 i. e. the isoelectric point of the fraction is 5.2 of pH. This value of the isoelectric point of the lens protein is coincident with the value reported already by Hesselvik. This indicates that this fraction is α-crystalline.

Fig. 4. Zone electrophoretic pattern of soluble protein of rabbit lens (Naphthalene cataractous whole lens).



Hight of initial zone 23 cm
Applied sample: immature 2.0 mg immature 3,0 mg mature 3.0 mg incipient
1/40 phosphate buffer pH 7.6 u=0.1 800 V 25 mA 17 hours
Each fraction 1.8 ml.

Investigation of the fraction with low mobility with the same procedure revealed that the isoelectric point of the second fraction could not be determined by this method. In reference to the previous reports, however, it seems evident that this fraction is β-crystalline.

Protein of bovine lens nucleus. The nucleus protein used for the experiment was prepared with care from the core of the lens after the removal of the lens capsule, the cortex and massa intermedia. Electrophoresis disclosed that the nucleus soluble proteins were made up of the two fractions, which was coincident with the electrophoretic pattern of the Spinco Analysis Model H as in the case of the lens cortex.

Differences are, that the fraction with high mobility is far less in quantity than the fraction with low mobility. This is the reason why the authors have been unsuccessful in ultracentrifugative analysis of this fraction. Complete analytical separation was not carried out with each of the two fractions of nucleus protein.

Ultracentrifugative analyses with the Spinco Analysis Model E were done with the protein solutions previous to zone electrophoresis, with which the nucleus soluble protein could not be fractionated into more than two fractions. Therefore, the authors are convinced that the soluble lens proteins are separated and purified into their components completely.

Studies were also made of precipitation reaction of each fraction of the nucleus protein at the isoelectric point as in the case of the cortex.

It was thus revealed, that the fraction with high mobility was α-crystalline with the isoelectric point of pH 5.2. The fraction with low mobility could not be precipitated at any pH, showing that the second component might be β-crystalline.

Electrophoretic patterns of cortical and nuclear soluble protein simultaneously fractionated by means of two separate starch columns. Modification of the technique was doneas follows: each of the two starch columns is connected to the same buffer solution tube. Then each starch column is connected to a buffer solution tube on the side of the negative electrode by means of two union tubes. With this modification, electrophoresis of the two separate sample was carried out under the same condition at the time. It was clarified with this modification that α-crystalline of both the cortex and the nucleus had the same mobility. The same is the case with β-crystalline. This seems reasonable because α-crystalline of the cortex and nucleus had been shown to have the same isoelectric point. Correspondence in mobility of β-crystalline of the cortex and the nucleus shows, that electrophoretic behaviour of these two proteins is the same or resembles one another.

Electrophoresis was again performed with the mixture of each of the two fractions of the cortex and the nucleus by means of the modified apparatus. It was thus revealed that each of proteins of two compoments of the cortex and the nucleus were made up of a single component.

II. Studies on the Soluble Protein of the Rabbit Lens

There is no doubt, that preparation of respective lens protein fractions of normal rabbit eyes in the purest and native state is a prerequisite to the clarification of changes in the lens protein fractions of cataractous eyes. As was mentioned in the previous chapter, the bovine lens proteins were found to be separated into their fractions in pure forms with relative ease. Thus, this analytical technique was applied to the separation of the soluble lens proteins of normal and naphthalene cotaractous rabbit eyes with the following results.

1) Soluble Lens Proteins of Normal Rabbit Eyes

Results obtained in the 1/10M veronal-HCl buffer system. Zone electrophoresis was performed with soluble lens proteins of normal rabbits in the 1/10 M veronal-HCl buffer system. Typical electrophoretic patterns with two peaks of fractions are obtained from the total lens, cortex and nucleus, respectively.

Fractions with high mobility available from the cortex and nucleus were precipitated at pH 5.0 by the addition of acetic acid, while fractions with low mobility were not obserevd to precipitate at any pH value. Therefore, it seems reasonable to assume, that the former

is α-crystalline and the latter β-crystalline.

Results in the 1/40 M phosphate buffer system. Electrophoretic patterns were obtained with the modification of the buffer system from the 1/10 M veronal-HCl buffer to the 1/40 M phosphate buffer system (1/40 KH_2PO_4-K_2HPO_4 pH 7.6 μ=0.1). It was revealed that the soluble proteins were separated into three fractions in the phosphate buffer in spite of the fact that two fractions had been found to exist when fractionated in the veronal-HCl buffer system. This shows that one of the two fractions in the veronal-HCl buffer system is further fractionated into two fractions in the phosphate buffer system.

With this problem in mind, the following experiment was performed. An attempt was made to separate each contents of the test tubes with the highest concentration of the respective fractions obtained in the veronal-HCl buffer system.

As results unsymmetrical patterns were obtained of β-crystalline of both the cortex and the nucleus, but not any new electrophoretic pattern was obtained from both the cortex and the nucleus.

Dialysis of the material was performed for 24 hours in pure water previous to the electrophoretic analysis. Electrophoresis of the supernatant was done in phosphate buffer after centrifugation with 6,000 r.p.m. for 1 hour. The third fraction with the lowest mobility was observed to decrease in the protein solutions from both the cortex and the nucleus. This fraction may belong to β-crystalline which is euglobulin hardly soluble in water. The first and the second fraction is under investigation (The later experiment revealed that the first fraction belonged to α-crystalline, the second and third to β-crystalline.).

2) Naphthalene Cataractous Lens Protein

Naphthalene suspended in rapeseed oil was given orally in a dose of 1.5 g. per kg. body weight with the help of a gastric tube every day or every other day. Both eyes of the rabbits which developed cataract were enucleated and used as materials. As the cataract progressed, the second fraction of soluble protein of the total lens was observed to decrease and the third fraction showed the smallest decrease, when separated in the 1/40 M phosphate bufter system.

The same is the case with the experiment with the cortex and the nucleus. It was found that in naphthalene cataract the second fraction begins to decrease already in the early stage of cataract and disappears completely in the state of mature cataract. On the other hand,decrease in the first fraction is gradual and the third fraction makes a slight decrease in the mature stage of cataract.

Conclusion

By means of an apparatus of zone electrophoresis on the starch column of vertical type, experiments were carried out with bovine and rabbit lens proteins separated in reference to Fromageot's method. Investigation of these protein solutions revealed the following results:

1. Electrophoretic patterns of soluble lens protein with this electrophoresis apparatus produce the same results as in the case of the Spinco Analysis Model H.

2. When electrophoresis is performed in the 1/10 M veronal-HCl buffer system (pH 8.6, μ=0.1) bovine and rabbit soluble lens proteins obtained from both the cortex and the nucleus are separated into α-and β-crystalline.

3. α-and β-crystalline are separated from the protein solutions of the cortex and the nucleus in their pure forms with this apparatus, which is under investigation with the Spinco Analysis Model E.

4. α-crystalline of the cortex and of the nucleus obtained from the bovine lens is observed to have the same isoelectric point of pH 5.2.

5. α-crystalline and β-crystalline separated from the lens cortex and the nucleus of rabbit and boine eyes shows the same electrical behaviour in the veronal-HCl buffer system, respectively.

6. Three soluble protein fractions are found to exist when electrophoresis of the total lens, cortex and nucleus proteins of rabbit eyes is carried out in the 1/40 M phosphate buffer system (pH 7.6 μ=0.1). The third fraction of this electrophoretic pattern is considered to be β-crystalline.

7. In the lens of naphthalene cataractous rabbit eyes the second fraction is observed to decrease already in the early stage of cataract and disappears completely in the mature stage. On the other hand, the first fraction decreases gradually as the cataract progresses.

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Editorial News

The twelfth Clinical Ophthalmological Congress was held at the Tokyo University on November, 9, 1958

The meeting was a great success, attended by more than seven hundred ophthalmologist.

A special speach was made by Dr. Shin-ichi Shikano, assist. Prof. of Tokyo University, about the pathological findings of Behçet's Syndrome

The number of general speaches amounted to more than fifty. The discussions were focused mainly on amblyopia, contact lens, EKC, trachoma and Behçet's disease.

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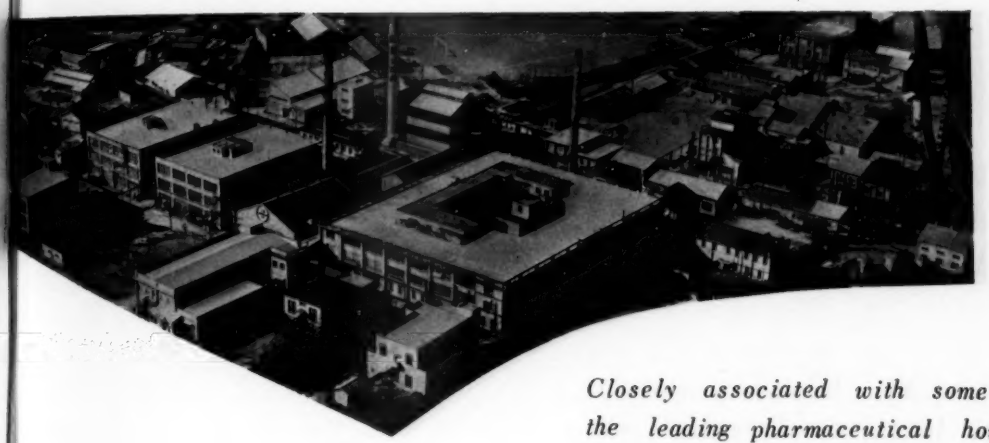

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